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DISTURBANCES
OF THE
KIDNEY

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DISTURBANCES OF THE KIDNEY

The Diagnosis and Treatment of the Various Disorders of the Kidney and Urinary Tract

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PREFACE

This series of articles deals primarily with the therapy of kidney disturbances, though the intimate connection between scientific therapy and the anatomy, physiology, function and pathology of the organs has demanded a fairly comprehensive review of these phases of the subject. The articles first appeared in the Therapeutics Department of THE JOURNAL. They have been slightly revised and extended. Numerous requests for reprint in convenient book form have prompted the present publication.

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ANATOMY, PHYSIOLOGY AND FUNCTION

In order to treat an abnormal or diseased organ correctly, it is essential that its structure and its function be understood. Hence the discussion of the various disturbances of the kidneys will include such data as will afford a rational basis for the treatment.

ANATOMY AND PHYSIOLOGY OF KIDNEYS

The kidneys are extraperitoneal, and lie normally on each side of the spine at the level of the twelfth thoracic vertebra and the first, second, and sometimes part of the third lumbar vertebra. The right kidney is about half an inch lower than the left, and is the one most frequently displaced. An average normal kidney is nearly 5 inches long, 3 inches wide, 1 inch thick, and weighs between 4 and 5 ounces.

The location and the approximate size of the kidneys may sometimes be determined by manual palpation with the patient lying on his back with the knees flexed. He is instructed to breathe slowly and deeply. By this method the lower extremities of the kidneys may be felt, provided the abdomen is not too fat or too distended. If a kidney can be depressed to the umbilical line and felt to slip back from the fingers again, it is movable. If it can be actually displaced, it is floating. A floating kidney may have had no normal attachments from birth, in which case its only support is the mesenteric.

Frequently even a floating kidney cannot be found or diagnosed, unless the patient is standing, partly supporting himself by resting his hand on the back of a chair, with the foot of the side under examination resting on the lower round of the chair. Even by this method a loose or floating kidney may not be discovered at a single examination, several being necessary before it is decided that the kidney is not movable, as the gas pressure in the intestines often holds a loose kidney in place. If a loose kidney is suspected, the patient, sitting up or standing, breathes rapidly and deeply, or jumps, and the loose kidney may be displaced downward. It can then be better palpated.

If a kidney is much enlarged, a dull percussion note will be found in a part of the abdomen which is ordinarily tympanitic. The right kidney is more frequently movable than the left, and may be mistaken for a distended pendent gallbladder; but if the kidney is floating, it can be moved much farther downward than the gallbladder could ordinarily be displaced. The most frequent causes of an enlarged kidney are hydronephrosis, pyonephrosis, tuberculosis, sarcoma, carcinoma, and echinococcus and cystic disease. Abscesses or growths behind and above a kidney may also displace it, and, if they are adherent to the kidney, the latter may seem enlarged.

The kidneys, like the liver and spleen, although not to such an extent, can change their size from time to time, depending on their blood supply. They may also grow large permanently in those who drink alcoholic beverages habitually and who require their kidneys constantly to do an increased amount of work.

The blood supply to the kidneys is large. It has even been estimated that under high blood pressure or under the action of diuretics the amount of blood passed through the kidneys in a minute may equal the weight of the kidney. Councilman[1] estimated that the blood supply to the kidneys may vary nineteen times as much as the average supply to other organs.

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[1] Councilman, W. T.: The Pathology of the Kidney, THE JOURNAL A. M. A., Jan. 13, 1906, p. 81.

In younger life the kidney structure may rapidly undergo repair even after considerable damage, but this ability to regenerate diminishes as age advances.

FUNCTION OF KIDNEY

The kidney structure is divided into a varying number of closely connected lobes (from eight to ten), each having a cortical and a medullary portion. The drainage system of the kidney consists, according to Gerrish, of about half a million capsules and about 15 miles of tubes variously termed as each tube proceeds from the capsule to the kidney pelvis, the constricted, the convoluted, the twisted, the looped (Henle), the zigzag, the second convoluted, the arched, and the long straight portion. All of these tubes are lined with epithelial cells, their shape and contour varying with the different parts of the tube. Tufts of capillaries in the capsules make up the structures termed the malpighian bodies, which are all situated in the cortex of the kidney. Each tube is also surrounded by a network of blood vessels.

There is a large surplus, as far as the body need is concerned, of secreting and excreting substance of the kidney. Not only may one kidney be removed (the other being normal) without damage to the health and general metabolism of the individual, but also it has been estimated that even as little as one fourth of the combined kidney substance (one half of one kidney), if healthy, is able to carry on necessary excretion. This explains why a person may live with apparently so much kidney structure diseased. However, the single kidney or the healthy parts of otherwise diseased kidneys must and do work with increased energy. This increased energy can be maintained only by a perfect blood supply to the healthy section of the kidneys; hence impairment of the general or local circulation is immediately serious. Any swelling of the epithelium that will narrow or block the tubules in the healthy section of these damaged kidneys, or any blocking by pressure from kidney edema, is serious. With a minimum amount of healthy kidney structure, any poison or substance irritating the kidneys is dangerous, and a drug ordinarily harmless — for instance, arsenic — may by its action on the kidney epithelium in damaged kidneys be fatal. Therefore, before arsenic is administered in large doses, as in syphilis, it must be ascertained that the kidney function is not seriously impaired.

It will be recognized that the pressure in the renal arteries must be sufficient always for the circulation of blood through the vessels in the kidney structure and for the proper excretion of urine and for the proper purity of the blood in the renal veins. The pressure on the inside of the capsules of the glomeruli must be lower than that of the circulation. Hence any serious damming back of urine will interfere with excretion.

Normal functioning of several of the internal secreting glands is essential for the complete functioning of the kidneys; perhaps more especially important is the secretion of the suprarenals as demonstrated by Marshall and Davis.[2] A damaged, insufficient heart must cause, sooner or later, imperfect circulation in the kidneys, imperfect excretion of urine, and, from the resulting passive congestion, diseased kidney structure.

The renal nerves come from the renal plexus and small splanchnic, and some fibers from the pneumogastric. Asher and Pearce[3] believe that these fibers from the vagi are secretory to the kidney. However, all secretory nerves are supposed to originate in the sympathetic system. Stimulation of the renal nerves will cause constriction of the renal vessels and a diminished flow of urine; hence some incidental irritation from a nerve lesion or reflexes from some abdominal inflammation may interfere with the function of the kidney. If a renal nerve is paralyzed, the kidney will swell and the flow of urine will increase, provided the general circulation is normal. Polyuria may also be caused by cutting the splanchnic nerve.[4] The well known result of puncture of a certain portion of the floor of the fourth ventricle, namely, polyuria, shows a central nervous control of the secretion of the kidneys. Cushing's work on the pituitary has shown the relation of the hypophysis to kidney function. Injuries to the head, and some cerebral lesions, especially in the region of the medulla, may cause polyuria and at times even glycosuria. It has not been shown that the kidney itself [illegible] so affected as to cause a glycosuria in some disturbances of the thyroid and pituitary, though glycosuria may occur in disturbances of these glands. Pituitary stimulation of the kidney function has been repeatedly shown by Cushing and others. Motzfeld[5] has shown that the posterior lobe of the pituitary has a governing influence on the renal function, and insufficiency of this secretion causes diabetes insipidus.

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[2] Marshall and Davis: Jour. Pharmacol. and Exper. Therap., 1916, viii, 525.

[3] Asher and Pearce: Zentralbl. f. Physiol., 1913, xxvii, 584.

If the general blood pressure rises, and more especially if the surface vessels contract, as from cold, there is an increased flow of urine, provided the renal arteries are not overcontracted. If the renal arteries are dilated while the general arterial system is not, the flow of urine will be increased, provided the flow of blood in the renal vessels is unimpaired. With excessively low blood pressure, there is no flow of urine.

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[4] Jungmann: München. med. Wchnschr., Aug. 12, 1913.

[5] Motzfeld: Boston Med. and Surg. Jour., 1916, clxxiv, 644.

The amount of kidney excretion is also much influenced by the changes in the blood composition. For this reason certain drugs may act as diuretics besides those which stimulate the heart and raise the blood pressure. Urea, dextrose, sodium chlorid and various salts act as diuretics. The reason some salts increase the flow of urine is probably that they raise the specific gravity of the blood, which then seeks water from the tissues to become isotonic with fluids outside of the blood vessels. This increased water in the blood is soon eliminated by the kidneys, and carries with it more or less of the extra salt. Caffein stimulates the nervous system, raises the general blood pressure, and soon generally causes diuresis by this circulatory effect alone. The first effect of caffein on the kidney vessels is to constrict them, but this is soon followed by a dilatation of these vessels.

The great value of digitalis as a diuretic is in cardiac insufficiency. If the systemic circulation is improved and if the kidneys are not too much damaged in this disease, diuresis will occur following digitalis administration. Thyroid extract may stimulate kidney secretion, as well as prevent poisoning from retained toxins in the blood in kidney insufficiency. Not only may glucose act as a diuretic, but small doses of saccharose, as 1 c.c. of a 100 per cent. solution in water, injected subcutaneously causes diuresis.[6]

GLAND ACTION

The flattened epithelial cells lining the capsules of the glomeruli take water and salts out of the blood and then excrete them, and their activity is largely dependent on the activity of the circulation in the malpighian tufts of capillaries, though, as just suggested, they may be stimulated to greater activity by certain substances. This excretion, therefore, is both active and passive. By passive excretion is understood the filtration due to circulatory pressure. This excreted water is then passed into the urinary tubules.

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[6] Crispolti: Policlinico, Rome, May, 1915, Med. Section.

The rodded cells of the convoluted tubules and ascending loop of Henle excrete urea and uric acid and some abnormal constituents of the blood, with a certain amount of water, their activity perhaps being determined by the amount of uric acid and urea in the blood. Also the tubules and especially the convoluted tubules excrete the sulphates and phosphates. The kidney epithelial cells in activity and at rest show different microscopic appearances, namely, in the granules.

Water exists in the protoplasm in from 70 to 75 per cent., and is continuously being absorbed and continuously excreted to keep the amount at normal. Atwater, who did so much careful experimentation on the excretion of water, considered that in twenty-four hours, with a normal subject at rest, about 1,000 c.c. of water were excreted in the urine, 1,000 c.c. by the lungs, 500 c.c. by the skin, and 250 c.c. by the bowels, making a total daily excretion of about 2,750 c.c. Abstinence from water tends to cause an increase in the specific gravity of the blood plasma in spite of the blood taking water from the tissues. The ability of the blood plasma to dissolve, transport and excrete salts also becomes diminished. Owing to this privation of water, the oxidation of foods, general metabolism and the absorption of muscle waste products soon become progressively imperfect. In other words, a certain amount of water is absolutely necessary for proper metabolism and excretion.

On the other hand, if an excess of water is retained in the blood and tissues, the specific gravity of the former may be lowered and a condition of hydremia occur. The first effect of this overfilling of the blood vessels is that more labor is thrown on the heart. If the heart is sufficient and the kidneys are perfect, unless there is exertion and profuse sweating, the extra water will be excreted by the kidneys. If the heart is insufficient or the kidneys are insufficient, edema and dropsy will generally occur. It should also be noted that water can be retained in the body without evident edema, an internal edema, so to speak, but frequent weighing of the patient will determine this fact.

In this hydremic tendency of the blood, nutrition of the cells is interfered with, water is not properly absorbed from the intestine, and nutriment is not well digested or absorbed and utilized.

Bogert, Underhill and Mendel[7] have shown that, in nephritis, water may be retained in the circulation in an abnormal amount. Hence the circulation is overtaxed until the extra water is gradually pushed through the impaired kidneys. They think that in nephritis there is an arterial and capillary wall disturbance which prevents normal osmosis.

This consideration of water metabolism is therapeutically interesting. When the urine is diminished in amount, of high specific gravity, the circulation perfect, the kidneys not badly defective and there is no edema, more water should be ingested. In weakened conditions of the circulation with edema, and in serious kidney defects, the amount of water ingested should generally be diminished. When the skin is dry and there is almost shriveling of the tissues on account of a large excretion of water, with the circulation good, and without edema, water should be given in large amounts. In other words, it is wrong to give large amounts of water in cardiac weakness; it is wrong to give large amounts of water in serious kidney defect; it is generally wrong to withhold water in diabetes, and the amount of water prescribed in gouty or other chronic disturbances depends on the condition of the heart and kidneys. Water should generally be given freely in acute feverish processes, unless there are edemas—especially of the lungs—as it helps to relieve kidney congestion, and also helps to eliminate the toxin. After chloroform and ether narcosis, colon injections of physiologic sodium chlorid solution may be given to aid the function of the kidneys, and many albuminurias and renal congestions will be avoided.

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[7] Bogert, Underhill and Mendel: Am. Jour. Physiol., 1916, xli, 189, 219, 229.

If the urine is acid, it may be inferred that the blood is normal or subalkaline. If the urine is alkaline, it may be inferred that the blood is strongly alkaline.

The odor of urine has been supposed to be due to a combination of volatile substances. Dehn and Hartman[8] have found a substance which they call "urinod" which they assert gives to urine its characteristic odor. It occurs in all urine in the minute amount of 1 to 2 parts in 100,000 parts. When the urine is fresh, the odor is blended with other compounds. The odor varies in intensity, depending on whether the urine is acid, neutral or alkaline. The odor of pure urinod is offensive, and is brought out by alkaline evaporation and by fermenting the urine.

The urine contains the starch-digesting amylase. If amylase is decreased in amount, either the kidney function or the ability to excrete amylase is impaired, or amylase may not be produced in normal amounts from the salivary and pancreatic glands, and hence less reaches the blood. Feeding amylase or glands does not ordinarily increase the amylase of the urine.

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[8] Dehn and Hartman: Jour. Am. Chem. Soc., 1914, xxxvi, 2136.

Normally the skin may excrete a bare trace of urea. If the kidneys are normal, the intestine seems to excrete but little urea.[9] This does not prove that, when the kidneys are diseased, the intestines could not help in the excretion of urea.

The kidneys normally eliminate iodin rapidly, nearly 80 per cent. in the first twenty-four hours after a single test dose, and most of the remainder in the next twenty-four hours. Even in an acute or chronic diffuse nephritis, iodin may be eliminated in nearly normal amount and speed, the amount eliminated in the urine bearing no relation to the amount of urine passed; but in interstitial nephritis the amount of iodin eliminated in twenty-four hours is greatly decreased, and it may take five or six days before it is all eliminated. A test dose is 1 gram of potassium iodid.[10]

While the vegetable acids, such as citrates, burn to alkali in the body, the tartrates are not so converted, and leave the body nearly in their original form. Underhill[11] and others have shown that tartrates in large doses can cause tubular nephritis in animals. While human beings tolerate without apparent kidney disturbance small doses of tartrates, either given medicinally or as occur in baking powders and in certain foods, and while it would probably require very large doses to cause kidney inflammation, it would seem inadvisable to give food rich in tartrates or to give medicinally large doses of tartrates in nephritis.

The amount of sugar in the blood varies from 0.04 to 0.12 per cent. with an average of 0.084 per cent., and with carbohydrate in the food the amount of sugar will be increased one hour after such a meal.[12] This emphasizes the advisability of seeking for a dietetic glycosuria in from two to three hours after a carbohydrate meal.

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[9] Van Slyke, D. D.; Courtney, A. M., and Fales, H. L.: Forms of Nitrogen in the Stools of Infants, Am. Jour. Dis. Child., June, 1915, p. 533.

[10] Sivert and Kaminsky: Russk. Vrach, 1914, xiii, No. 20.

[11] Underhill: Jour. Exper. Med., 1913, xviii, 317, 322, 347; Jour. Biol. Chem., 1912, xii, 115.

TESTS OF KIDNEY FUNCTION AND EFFICIENCY

Geraghty and Rowntree[13] first presented the phenolsulphonephthalein test of renal function in 1910. Since that time, this and other tests for renal excretion have been perfected and described, and now a more or less accurate determination of the deviations from the normal of kidney excretion is easily made. The importance of determining the functional ability of the other kidney in questions of surgical interference with one of them, and of determining the functional ability of both for prognosis and for treatment in chronic nephritis, must be generally recognized. In ordinary disease one or more of the simpler tests should always be made.

The possibilities of elaborate tests and determinations have been noted by Rowntree.[14] The most commonly used tests are the phenolsulphonephthalein, potassium iodid, lactose, and diastase excretion tests, and urea and sodium chlorid retention tests. Other tests for color excretion besides the phenolsulphonephthalein are used, but are not so valuable. In patients under observation, the phenolsulphonephthalein test, as well as tests for retention of chlorids, iodid and perhaps lactose, should be repeated at intervals to indicate the progress of the disease and also the rate of improvement. Kidney excreting tissue can regenerate, a local lesion or passive obstruction may be removed, and the tests will indicate that this has occurred. Rowntree says that failure to detect diastase in the urine by the customary technic shows more or less serious renal injury.

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[12] Strouse, Stein and Wiseley: Bull. Johns Hopkins Hosp., June, 1915, p. 211.

[13] Geraghty, J. T., and Rowntree, L. G.: Jour. Pharmacol. and Exper. Therap., July, 1910, p. 579.

[14] Rowntree, L. G.: Am. Jour. Med. Sc., March, 1914, p. 352.

As tests of urinary excretion are more frequently made, there will be fewer unforetold uremic deaths. Kidneys which cannot well excrete phenolsulphonephthalein or lactose show insufficiency and the future probability of uremia. On the other hand, Rowntree cautions that occasionally the excretion of phenolsulphonephthalein may be normal, even with a positive nephritis. In combined heart and kidney insufficiency, these tests may decide how much the kidneys are at fault. In passive kidney congestion, though the excretion of lactose, iodid and salt may be delayed, Rowntree finds that the phenolsulphonephthalein excretion may be normal. Long continued passive kidney congestion from a defective heart must sooner or later, of course, lead to a chronic nephritis. If in combined heart and kidney disturbance the phenolsulphonephthalein test shows a greatly diminished secretion, even after the treatment has improved the heart, the prognosis of the kidney condition is bad.

Uremia may be present with a fair renal function, and recovery take place; on the other hand, even when the symptoms are mild, tests of excretion and of the blood may give a very grave prognosis, and the outcome will be fatal.

For general use for diagnosis and prognosis, Rowntree believes, and other observers agree, that the phenolsulphonephthalein test is the best. When this test is combined with a blood test for nonprotein nitrogen retention, the results are of great service. Iodid and salicylate tests are not of great prognostic value. Lactose is unreliable, according to Rowntree, because it is at times suppressed in moderate lesions, though in chronic nephritis its suppression is serious. If it is suppressed and the phenolsulphonephthalein output much diminished, uremia is impending.

A suppression of sodium chlorid indicates the necessity for a salt-free diet. The diastase test is often of interest, but is not reliable in determining the actual condition of the kidneys.

The foregoing tests are essential because one or two estimates of the urea excretion in the twenty-four hour urine (as previously relied on) may show a good amount of excretion even with serious kidney disease, though a persistently low urea excretion (found in a series of examinations) is a serious omen. Hence a single twenty-four hour specimen of urine, in operations without emergency, should not be relied on by surgeons as determining the ability of the kidneys to withstand the ether, operative shock and postoperative disturbances. Functional tests alone are not of absolute significance when the kidneys are suffering from some condition other than nephritis, as such disturbance may greatly impair their excretion at a given period, but may allow more or less recovery in a shorter or longer time. Whenever the excretion of phenolsulphonephthalein is decreased, the amount of nonprotein nitrogen of the blood should be ascertained.

Christian[15] calls attention to the fact that a patient may properly excrete nitrogen, but have sodium chlorid retention on the least provocation. Such a condition should not be termed nephritis, but a disturbed salt elimination. However, these patients may later show also a lowered nitrogen excretion and develop nephritis.

It should be recognized that as yet it is impossible to decide from functional tests during life the exact pathologic condition of the kidneys which will be found on necropsy. As pointed out by Christian, even with a good output of phenolsulphonephthalein and a normal blood nitrogen, incipient chronic nephritis should be diagnosed when the following symptoms are present: slow excretion of sodium chlorid; a tendency to fixation of the specific gravity of the urine; periods of lessened urinary excretion and periods of polyuria; unexplainable attacks of fatigue; even a slight albuminuria, and cylindroids in the urine or an occasional hyaline cast. With the beginning of the disease thus determined, the physiologic tests of excretion will show the progress and the prognosis from month to month. A patient should not be worried by too frequent tests, though his diet should from time to time be arranged to conform to his ability to excrete the various elements of food and drink. This is especially true of the salt and water ratios of intake to excretion. Even without edema, water may irritate the kidneys, or, on the other hand, it may be of benefit in promoting the excretion of salts, especially when the urine shows a more or less fixed low specific gravity.

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[15] Christian: Am. Jour. Med. Sc., May, 1916, p. 625.

As long urged by some clinicians, a low protein diet may be needed by the patient, but many a nephritic has been weakened and injured by protein privation.

Christian does not believe that the dyspnea of nephritics is generally an acidosis, and administration of large amounts of alkalies does not often remove the dyspnea. However, Palmer and Henderson[16] believe that severe injury to the glomeruli results in reduced ammonia excretion and at times causes an acidosis.

After several years of experience, Thayer[17] states that when the phenolsulphonephthalein test has been carefully made and the phenolsulphonephthalein is found not well excreted, postmortem examinations of the kidneys have always shown nephritis. He also states that in gradually developing uremia, the output of phenolsulphonephthalein generally diminishes to the vanishing point during the last days of the patient's life.

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[16] Palmer, W. W., and Henderson, L. J.: A Study of the Several Factors of Acid Excretion in Nephritis, Arch. Int. Med., July, 1915, p. 109.

[17] Thayer: Am. Jour. Med. Sc., December, 1914, p. 781.

On the other hand, when a kidney is in such a condition that it must be surgically removed, the other kidney, owing to reflex disturbance, may not perfectly excrete the phenolsulphonephthalein, and yet after the offending kidney has been removed the remaining kidney may greatly improve and even normally excrete phenolsulphonephthalein. Less than 20 per cent. of phenolsulphonephthalein output in the first two hours is a danger signal, but even a much larger output must not be considered as evidence of operative safety, unless there is no nitrogen retention in the blood, and other signs of uremia are absent.[18] It should be emphasized that considerable irritation in either one or both kidneys, even from a simple pelvic calculus, may prevent proper excretion of phenolsulphonephthalein, even without nephritis. The same is true in hydronephrosis, tuberculosis of the kidney, or other tumor of the kidney. Not only is the output of the affected kidney impaired, but that of the other may also be diminished.

It is self-evident how essential it is in all cases of possible operative interference in patients with impaired kidney function to obtain specimens of urine from each kidney, and also, if possible, separate specimens after the phenolsulphonephthalein injection. G. M. Smith[19] suggests that after ureter catheters are in place the patient be given an intravenous injection of 0.006 gm. of phenolsulphonephthalein in solution. Each normal kidney should begin to excrete it in three minutes, and in fifteen minutes should excrete 15 per cent. of it. If the well kidney secretes more than 15 per cent. in that length of time, he believes it shows that it has assumed part of the work of the diseased kidney.

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[18] Braasch, W. F., and Thomas, G. J.: Practical Value of Chemical Tests of Renal Function in Surgical Conditions of the Urinary Tract, JOURNAL A. M. A., Jan. 9, 1915, p. 104.

[19] Smith, G. M.: Separate Renal Function, THE JOURNAL A. M. A., Jan. 16, 1915, p. 223.

Besides the usual estimation of urea excretion in twenty-four hours and the phenolsulphonephthalein test, Stevens[20] suggests a rapid phlorizin test of each kidney. His technic is described in his article.

In acute arsenic or mercuric chlorid poisoning, the phenolsulphonephthalein test will give an early indication of involvement of the kidneys.

TECHNIC OF PHENOLSULPHONEPHTHALEIN TEST

Aseptic ampules prepared for subcutaneous injection can be obtained,[21] each ampule containing 1 c.c. of sterile salt solution and 0.006 gm. of phenolsulphonephthalein. Half an hour before the injection the patient should be given 250 c.c. of water to insure that urinary secretion will not be delayed on account of a shortage of water. Next, the patient is catheterized under aseptic precautions, or he empties his own bladder. Then the phenolsulphonephthalein is given subcutaneously or intramuscularly, or rarely intravenously. When it is given subcutaneously or intramuscularly, the patient should urinate at the end of the first hour and again at the end of the second hour, into different receptacles; or if a catheter is in the bladder, the urine may drip into a test tube containing a drop of a 25 per cent. solution of sodium hydroxid, and the first slight pink color should be noted and the time recorded. The catheter is then clamped and the urine drawn at the end of the first hour, and again at the end of the second hour. Each sample of urine should be measured and tested for its specific gravity; then enough of a 25 per cent. solution of sodium hydroxid is added to the urine to make the color which in acid urine is yellow or orange change to the deepest possible reddish purple. This solution is diluted with distilled water to 1,000 c.c., and the whole thoroughly mixed. A small portion is filtered and placed in a color test glass for comparison with some standard or colorimeter, of which several types may be obtained.

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[20] Stevens, W. E.: The Comparative Value of Modern Functional Kidney Tests, The Journal A. M. A., May 16, 1914, p. 1544.

[21] See New and Nonofficial Remedies, 1916, p. 247.

It should be noted that the phenolsulphonephthalein may be freely excreted though but a small amount of urine is passed, that is, the amount of urine passed has no relation to the amount of phenolsulphonephthalein excreted. Normally, on the average, 50 per cent. should be recovered in the first hour, and 85 per cent. at the end of two hours, from intramuscular injections. From intravenous injections, from 35 to 45 per cent. should be recovered the first fifteen minutes, from 50 to 65 per cent. in thirty minutes, and from 65 to 85 per cent. in one hour. If in two hours, when it is given intramuscularly, only 40 per cent. or less of the phenolsulphonephthalein has been excreted, Elliott[22] considers that not only are the kidneys defective, but also there is a retention of waste nitrogen in the blood, and a blood test should be made.

To note just when the elimination of phenolsulphonephthalein ceases, the patient may pass the urine every hour for six hours.

OTHER TESTS OF FUNCTION

In the iodid test of von Noorden,[23] a capsule containing 0.2 gm. of potassium iodid is given on an empty stomach, swallowed with 50 c.c. of water. The saliva is tested for iodin every three minutes to show when the capsule dissolves, and the urine is drawn and tested every five minutes. Normally the saliva shows iodin in from eight to twelve minutes, and the urine shows it from three to five minutes later. If the reaction in the urine is delayed half an hour, it is a moderate delay; if it is delayed from this time to one hour, the delay becomes more and more significant. All the iodin should be eliminated in thirty hours.

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[22] 22. Elliott, A. R.: The Phenolsulphonephthalein Test in Chronic Nephritis, The Journal A. M. A., June 5, 1915, p. 1885.

[23] 23. Von Noorden: Med. Klin., 1916, xii, No. 1.

Thomas[24] urges the use of indigocarmin as a valuable indication of the functional capacity of each kidney, as evidenced by the output of colored urine at each ureter during ureteroscopy. He finds it valuable in all surgical conditions, and especially valuable in conjunction with roentgenoscopy in the diagnosis of renal calculi. After the patient is prepared, according to the technic which he describes, 20 c.c. of a 0.4 per cent. solution of indigocarmin are injected into the gluteal muscles, and if the kidneys are normal, they will excrete a dark blue liquid in from three to twenty minutes and a light blue in fifteen minutes. In his 200 tests it appeared as light or dark blue in fifteen minutes in 90 per cent. of the cases, and in ten minutes in 61 per cent. of the cases. If the indigo does not appear as light blue in fifteen minutes or as dark blue in twenty minutes, there is a serious insufficiency of renal function. Of course the ureteroscope will show in surgical conditions a more or less marked discrepancy in the excretion from the two kidneys.

In a later article, Thomas[25] emphasizes his belief that his low mortality in prostatectomy (3.3 per cent.)

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[24] Thomas, B. A.: The Results of Two Hundred Chromo-Ureteroscopies Employing Indigocarmin as a Functional Kidney Test, The Journal A. M. A., Jan. 18, 1913, p. 185.

[25] Thomas, B. A.: The Rôle of Functional Kidney Tests and Preoperative and Postoperative Treatment in the Reduction of Prostatectomy Mortality, The Journal A. M. A., Nov. 28, 1914, p. 1909.

is due to the previous indigocarmin test and a recognition of operative limitations. Although this test to decide the difference between a surgical kidney and its fellow is of value, as described by Thomas, it is not as good a test of renal gland function as is the phenolsulphonephthalein test, as only 25 per cent. of indigocarmin is excreted by the kidneys. Furthermore, indigocarmin excretion may not be decreased in some instances of acute or diffuse nephritis, and a blue color may not develop in purulent and alkalized urines.[26]

The methylene blue test is not as good as the phenolsulphonephthalein test, and has the same defects as the indigocarmin.[27]

Neubauer[28] gives 1.5 gm. of creatinin in 150 c.c. of sweetened water, and finds that healthy kidneys eliminate from 60 to 90 per cent. of it in six hours and the remainder in six more hours. With diseased kidneys less than 20 per cent. is eliminated in six hours, and the rest not in days. While in nephritics lactose excretion is always delayed, with serious congestion of the kidneys, its excretion does not follow the rate of phenolsulphonephthalein excretion and does not show what part of the kidneys is affected.[27]

In a recent article, Mosenthal and Lewis[29] state that too much reliance must not be placed on single tests of renal function, as kidneys may retain sodium chlorid, urea, phosphates, sulphates, or even water — any one of these substances — and yet allow the remaining substances to be excreted. They place great value on the test meal for kidney function, on phenolsulphonephthalein, and on Ambard's formula.[30] After a study of the subject, however, Jonas and Austin[31] conclude that the estimation of the blood urea is more reliable and more useful than is the urea index or the Ambard quotient.

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[26] Hepburn: Yale Med. Jour., March, 1912, p. 289.

[27] Potter and Bell: Am. Jour. Med. Sc., February, 1915, p. 236.

[28] Neubauer: München. med. Wchnschr., 1914, lxi, No. 16.

[29] Mosenthal, H. O., and Lewis, D. S.: A Comparative Study of Tests for Renal Function, THE JOURNAL A. M. A., Sept. 23, 1916, p. 933.

[30] McLean: Jour. Exper. Med., 1915, xxii, 212; Smith, THE JOURNAL A. M. A., Jan. 27, 1917, p. 278.

DETERMINATION OF THE NITROGEN OF THE BLOOD

The examination of the blood for the total nonprotein nitrogen and for urea, together with the tests of the kidney excretion described above, has advanced the understanding of kidney defects, and presages the limitations of medical and surgical treatment of genitourinary disease. The method of Folin,[32] in which from 2 to 5 c.c. of blood are required, is probably the test most used.

Nonprotein nitrogen is a term applied to the nitrogen remaining after all proteins have been precipitated out of the blood. Such remaining nitrogen is also termed "incoagulable nitrogen."

In the beginning it should be urged that a single determination of the nonprotein nitrogen of the blood is not conclusive, unless the amount is large, but a progressive increase from day to day or week to week shows danger of uremia, and uremia is not a condition caused by one poison, but by many.

Tileston and Comfort[33] believe that the nonprotein nitrogen of the blood below 30 mg. per hundred c.c. of blood is normal; from 30 to 35 mg. it shows a slight increase; from 35 to 50 mg. a considerable increase, and from 50 to 100 mg. a great increase. Nonprotein nitrogen of 100 mg. or more is of very serious prognosis. More than 16 mg. of urea per hundred c.c. of blood is abnormal, and more than 25 mg. shows a considerable increase. This agrees closely with the figures offered by Bang[34] of Sweden, who says that 25 mg. of nonprotein nitrogen in 100 gm. of blood is normal, and about 15 mg. of this should be urea. More recently Gettler and Baker[35] have examined the blood in thirty normal individuals three hours after a uniform breakfast, and find the nonprotein nitrogen higher than the foregoing figures, namely, from 30 to 45 mg., and the urea from 15 to 25 mg.

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[31] Jonas, Leon, and Austin, J. H.: Am. Jour. Med. Sc., October, 1916, p. 560.

[32] Folin, Otto: Jour. Biol. Chem., 1912, xi, 527.

[33] Tileston, Wilder, and Comfort, C. W., Jr.: The Total Non-Protein Nitrogen and the Urea of the Blood in Health and in Disease, as Estimated by Folin's Methods, Arch. Int. Med., November, 1914, p. 620.

Tileston and Comfort found that the determination of the total nonprotein nitrogen was more important than the urea determination. They found in chronic interstitial nephritis the nonprotein nitrogen increased even to 324 mg. per hundred cubic centimeters of blood, and the urea even to as much as 237 mg. Sometimes, however, there may be a normal nonprotein nitrogen in the forms of chronic nephritis.[36]

During acute disease, as pneumonia, there may be a retention of nitrogen in the blood, and such a condition is probably a frequent added cause of death in this most serious of acute diseases.

While uremic patients practically always show nitrogen retention, it is interesting to note that puerperal eclampsia does not always, and perhaps seldom, show such retention, unless there has been a long previous nephritis. In other words, puerperal eclampsia may or may not be a true uremia, as other retained intoxicants may cause convulsions besides those retained by kidney insufficiency.

The diet of a nephritic should be modified by the amount of nonprotein nitrogen of the blood. Surgical operations should be postponed if practical when the blood shows an increased amount of nonprotein nitrogen.

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[34] Bang: Biochem. Ztschr., 1915, lxxii.

[35] Gettler and Baker: Jour. Biol. Chem., 1916, xxv, 211.

[36] Frothingham, Channing, and Smillie, W. G.: The Relation Between the Phenolsulphonephthalein Excretion in the Urine and the Non-Protein Nitrogen Content of the Blood in Human Cases, Arch. Int. Med., October, 1914, p. 541.

As syphilis is now known to attack the kidneys more frequently than was supposed, and as retained nitrogen may be in evidence in this disease, the effect on the kidneys of arsenic and mercury treatment in these patients must be seriously considered. Whether or not the arsenic treatment predisposes to kidney disturbance later in syphilis should also be investigated. Acute intestinal obstruction has been shown to increase the blood nitrogen, and the same may be true of many acute painful or serious crises.

Ordinarily, but not always, in nephritis, the less the phenolsulphonephthalein output, the greater the amount of nonprotein nitrogen in the urine. According to Frothingham and Smillie,[36] and others, passive congestion of the kidneys from cardiac insufficiency may markedly diminish the output of phenolsulphonephthalein without an increase of the nonprotein nitrogen being found in the blood.

As shown by Hopkins and Jonas,[37] the amount of protein fed a chronic nephritis patient has a direct bearing on the amount of nitrogen retention, and therefore a frequent estimate of the nonprotein nitrogen of the blood has a distinct therapeutic value. Many nephritic patients are unnecessarily underfed. Mosenthal and Richards[38] came to the conclusion, after careful investigation in nephritic patients, that even when there was a retention of nitrogen in the body, as determined by the protein intake and the nitrogen output, there was not necessarily an increase in the nonprotein

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37. Hopkins, A. H., and Jonas, Leon: Studies in Renal Function with Special Reference to Non-Protein Nitrogen and Sugar Concentration in the Blood, Phenolsulphonephthalein Elimination and Blood Pressure, Arch. Int. Med., June, 1915, p. 964.

38. Mosenthal, H. O., and Richards, A. E.: Interpretation of Positive Nitrogen Balance in Nephritis, Arch. Int. Med., February, 1916, p. 329.

nitrogen of the blood. Hence at times in nephritics a normal protein nonprotein nitrogen in the blood may not show that nitrogen is not retained.

Cameron[39] says that after a general anesthetic there is usually an increase in the urea of the blood, and this is especially true in operations on the genito-urinary tract, and of course most frequently in patients who have a diminished kidney function.

Schwartz and McGill[40] give their findings after estimating the urea of the blood in 211 patients as follows: The urea of the blood is greatly modified by diet; is increased by nephritis, varying with the degree of inflammation and congestion; is increased by mechanical obstruction of the urinary tract; is generally increased in pneumonia; is increased in exophthalmic goiter; is decreased in myxedema, and in many acute conditions and infections it is increased.

Addis and Watanabe[41] state that the rate of excretion of urea by the urine varies with the urea concentration in the blood.

It was long in vogue to give large amounts of water in nephritis "to wash out the kidneys." More recently it was shown that this was bad in dropsy, in which the kidneys do not excrete water well. We must now come to the intermediate decision, namely, that with nitrogen retention and with damaged kidneys which cannot excrete a concentrated urine, and when there is no great amount of edema, we must give considerable water at the same time that we diminish the protein intake in order to eliminate the solids and retained nitrogen. The truth of this statement has recently been demonstrated by Foster and Davis.[42]

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[39] Cameron, D. F.: Variations in Renal Function Dependent on Surgical Procedures, The Journal A. M. A., June 3, 1916, p. 1765.

[40] Schwartz, Harold, and McGill, Caroline: Blood Urea Determinations in 211 Cases, Arch. Int. Med., January, 1916, p. 42.

[41] Addis and Watanabe: Jour. Biol. Chem., March, 1916, p. 1057.

[42] Foster and Davis: Am. Jour. Med. Sc., January, 1916, p. 44.

CREATIN AND CREATININ

Creatin is methyl-guanidin-acetic acid. Creatinin is an anhydrid of creatin.

Creatinin is a normal constituent of the urine, and the daily excretion is from 1 to 2 gm. It is both of exogenous and endogenous origin.

Many investigators have found that in nephritis there is retention of creatinin in the blood, and it has been found that if over 5 mg. of creatinin per hundred c.c. are retained, the outcome is fatal. The creatinin retention probably follows, and is coincident with the nonprotein nitrogen retention of the blood. It has been shown that there is a slight increase of the creatinin in the blood after a full meal, and in infections.

The creatin and creatinin content of the blood of children was recently investigated by Veeder and Johnson,[43] who examined seventy-five children in various stages of scarlet fever. They found that there is no apparent relationship between the amount of creatin and creatinin, but there is less creatin in the blood of children than in the blood of adults. On the other hand, creatin is found normally in the urine of children, but is not present in the urine of adults. Contrary to previous presumption, these investigators did not find any definite relation between the total nonprotein nitrogen and the creatinin-creatin content of the blood.

As stated, creatin is a normal constituent of children's urine, but is rare in the adult. The relation of the creatinin output to the amount of muscular tissue in adults is not the relation of the creatinin to the muscle tissue in children before puberty.

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[43] Veeder, B. S., and Johnston, M. R.: The Creatinin and Creatin Content of the Blood of Children, Am. Jour. Dis. Child., August, 1916, p. 136.

Cutler and Morse[44] have lately found in two children with nephritis no relation between the creatin and creatinin output and the amount of the fever.

Rose[45] and others, in experimental studies on creatin and creatinin, find that, contrary to the general belief, protein feeding in a starving man promptly reduces the creatin output to nothing. Administration of large doses of creatin in an ordinary individual causes some increase in the output of creatinin. This increase is doubtless due to the conversion of creatin intake to creatinin, and not to an increased output of endogenous creatinin.

EXPERIMENTAL NEPHRITIS

It cannot yet be definitely proved by tests of function exactly which parts of the kidney are pathologically affected. This has been shown recently by Frothingham,[46] though Potter and Bell[47] state their belief that an experimental tartaric acid nephritis shows that phenolsulphonephthalein, indigocarmin and methylene blue are excreted by the convoluted tubules. In acute glomerular nephritis caused by scarlet fever there is often a complete suppression of urine, that is, no water is passed by the kidneys. This bears out the belief that most of the water of urine is secreted by the glomeruli.

It has not been shown that the kidney has an internal secretion; it can, however, synthesize hippuric acid from glycocoll and benzoic acid, salicylic acid from salicylic acid and glycocoll. Uric acid has been shown to be excreted by the convoluted tubules and by the ascending loop of Henle, and urine leaving the Bowman capsule is alkaline, becoming acid in the convoluted tubules.

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[44] Cutter, I. S., and Morse, Max: Studies in the Metabolism of Children, Am. Jour. Dis. Child., May, 1916, p. 326.

[45] Rose, W. C., and Dimmitt, F. W.: Experimental Studies on Creatin and Creatinin, VII, The Fate of Creatin and Creatinin when Administered to Man, Jour. Biol. Chem., 1916, xxvi, 345.

[46] Frothingham: Am. Jour. Med. Sc., January, 1916, p. 72.

[47] Potter and Bell: Am. Jour. Med. Sc., February, 1915, p. 236.

Christian,[48] in summing up some practical results obtained from experimental nephritis, states that the administration of diuretic drugs, such as potassium acetate, caffein, theobromin sodium salicylate, theocin, and even water, shortens rather than prolongs the lives of these animals. Even when one or other of these drugs at first stimulates the excretion of urine, it soon lessens the excretion and does harm. Therefore not only should renal irritants not be given, but even renal stimulants should not be used in the therapy of acute or subacute nephritis. Large amounts of potassium salts, chlorids and iodids, and the substitution of potassium chlorid for sodium chlorid caused poisoning in the animals which were suffering from experimental nephritis. Hence such drugs must be dangerous to human beings who have acute nephritis, and even large amounts of water may do harm by first promoting excretion and then wearying defective kidneys. Therefore, as noted by Christian, after diuresis there is often a lowered output of nitrogen.

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[48] Christian: Am. Jour. Med. Sc., May, 1916, p. 625.

ABNORMALITIES OF THE URINE

A person weighing 50 pounds should normally excrete about 32 gm. of solids in the urine per day; one weighing 100 pounds, about 57 gm.; one weighing 150 pounds, about 76 gm., and one weighing 200 pounds, about 88 gm. Girls and women ordinarily do not excrete as large an amount, owing to a less active life. After the age of 40, the excretion generally diminishes progressively, in some cases becoming 30 per cent. less after the age of 60.

A simple method of computing the total solids in the urine is to take the specific gravity of the twenty-four hour specimen, multiply the total number of ounces of this specimen by the last two figures of the specific gravity, and add to it 10 per cent. of the figure thus obtained. The result will be the total amount of solids passed, provided the urine does not contain albumin or sugar.

With retention of chlorids in the body there is also a retention of water, but with the free excretion of chlorids, the water excretion is also increased.

The following is a discussion of conditions often not isolated and not demanding treatment, but nevertheless worthy of brief consideration.

POLYURIA: DIABETES INSIPIDUS

Transient polyuria is of frequent occurrence and may be due to nervous irritability or to nervous tension as seen in hysteria; in normal individuals, it may be due to some temporary nervous excitement. It may also be due to a sudden change of temperature; to the drinking of too much water, to an increased consumption of tea, coffee, or caffein; to a diuretic drug, or to intestinal indigestion. Polyuria may also be caused by fevers, by chronic hypertension, by cerebral injuries, by some growth or lesion in the brain, or by a pituitary disturbance.

Protracted polyuria, termed diabetes insipidus, is a peculiar condition causing the patient to pass gallons of urine per day of a specific gravity approximately that of water, namely, from 1.000 to 1.005. These patients suffer from an inordinate thirst, and unless large amounts of water are consumed, their tissues waste. They often suffer from vomiting, diarrhea or constipation, and headaches. If the patient is a child, growth is often stunted.

It was thought that the kidneys of these patients could not secrete urine of ordinary specific gravity, and hence to eliminate the solids an enormous amount of water was required. Patients apparently cured by treatment seem to disprove this hypothesis, and recently it has been shown that this condition is probably caused by a disturbance of the hypophysis cerebri. It has long been known that pathologic conditions in the brain cause polyuria. It is also known that pituitary extract stimulates the kidneys and at times acts as a diuretic, and that the posterior lobe of the hypophysis has some control over the kidney secretion. The relation between the pituitary and the kidneys has been described by Cushing in his book on the "Pituitary Body and Its Disorders." That a diminished secretion of the pituitary is a cause of diabetes insipidus seems to be borne out clinically, as some success has been reported in feeding posterior lobe extract to these patients.

At times modification of the circulation, raising the blood pressure when it is low and lowering it when it is high, will diminish polyuria. Bromids may act on the nervous system to correct polyuria; atropin has been successful at times; but in the real diabetes insipidus the most successful drug has been ergot, in fairly large doses and long continued. It is to be hoped that posterior hypophysis extract will be more successful.

In real diabetes insipidus, deprivation of water is impossible. The amount may be reduced somewhat, but the frequency with which water is needed cannot be reduced, and serious head and stomach symptoms may be the result of an attempt to limit the intake of water.

ANURIA

This condition may result from nephritis, from any serious condition of the genito-urinary tract, or reflexly in serious abdominal diseases. It may be the result of low blood pressure or of so-called hysterical conditions. Before anuria, or suppression, is diagnosed, it must be known that the bladder is empty and the ureters not obstructed.

A nephritic patient cannot live long with suppression; but in cases of nervous suppression, anuria may be a symptom for days.

If suppression is caused by uremia, the treatment is that of uremic poisoning. If there is obstruction, surgical interference of some kind is necessary. If the circulation is at fault, because of cardiac failure, digitalis and caffein may be indicated. If hemorrhage has occurred, and at times without hemorrhage having occurred, hypodermoclysis of physiologic sodium chlorid solution or a colon injection of salt solution may be given.

LOW ACIDITY OF THE URINE

This is a condition which seldom requires treatment. Urine which is neutral or close to neutral is not quite normal, except, perhaps, directly after a meal. The pelvis of the kidneys, the ureters, the bladder, and the urethra, under normal conditions, require that the urine be acid. Subacid or faintly alkaline urine has a tendency to deposit alkaline salts, to cause an increased secretion of mucus, and to irritate the bladder. Many pathogenic bacteria gain a foothold and grow in the parts bathed by this neutral or alkaline urine, which would not grow in acid urine. If there are symptoms of bladder irritability, and no other cause is ascertained than an alkaline or subacid urine, the patient should be allowed more meat unless it is otherwise contraindicated. He may also be given one of the following: dilute hydrochloric acid, hexamethylenamin or acid sodium phosphate. A gastric hyperacidity may be present, causing low acidity of the urine. This condition should be treated in the usual way by gastric sedatives and the administration of substances which inhibit acidity, such as olive oil, butter and atropin.

When on account of hyperacidity of the urine, or on account of acute irritation of the bladder or urethra, it is advisable to render the urine alkaline for a time, alkaline treatment should not be too long continued, as an alkaline urine may allow phosphate deposits on uric acid calculi, or decomposition and formation of ammonia in an already irritated bladder. The seriousness of chronic cystitis is, of course, recognized.

PHOSPHATURIA

If the urine is alkaline there may be a more or less constant phosphaturia, sometimes causing the passage of milky urine with a pinkish deposit more or less admixed with mucus on standing. Phosphaturia occurs in many nervous conditions, both organic and inorganic, at times, even in insomnia. In these cases all nervous excitement and mental irritation should be prevented, and sexual indulgence prohibited for a time.

The phosphates of these salts come mainly from the food, but are also derived from the breaking down of nucleic acid and proteins of the tissues. The phosphates are therefore increased in wasting diseases and in some forms of leukemia, but may be diminished in pregnancy when needed for the bones of the fetus.

A prolonged phosphaturia is a drain on the system, and there is always danger of causing deposits in the bladder, causing cystitis to develop. The same treatment should be given in phosphaturia as that suggested for diminished acidity of the urine, combined with any treatment required by the general condition of the patient.

INCREASED ACIDITY OF THE URINE

A hyperacidity of the urine may have no relation to the amount of uric acid excretion, but it is generally associated with a diminished bulk of urine, with high specific gravity, with a tendency to deposit, on standing, the amorphous urates with perhaps uric acid and oxalates. A deposit of "brick dust," so called, does not prove that such a sediment is largely uric acid.

An increased acidity of the urine and a too concentrated urine may cause backache; slight ureteral stinging pains, with or without the passage of an actual concretion; bladder irritability, due to irritation at the neck of the bladder, and an increased frequency of urination, with the passage of a small amount of urine. Such urine with the mucus and bacterial conditions right may deposit calculi in the kidney pelvis, or at times in the bladder.

Patients suffering from this disease should avoid meat (including fish and fowl), purin substances (tea, coffee, fish roe, etc.), and alcohol, and drink daily a large amount of pure water. Haskins[49] has recently shown the value of diluted urine in rendering uric acid as well as salts more soluble. This should be included in the treatment as well as some alkaline drug, as sodium carbonate, or perhaps better, sodium or potassium citrate in 2 gm. doses, three times a day. All mineral acids should be avoided, and all drugs that render the urine acid.

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[49] Haskins: Jour. Biol. Chem., 1916, xxvi, 205.

A modification of the foregoing intensive treatment may often be sufficient for relief and for temporary cure at least, but patients who have suffered from this increased acidity of the urine must for some time modify their diet and water drinking to prevent recurrence.

OXALURIA

The term "oxaluria" has been applied to the condition in which calcium oxalate crystals are deposited in urine after standing. This salt is largely held in solution by the acid phosphate of sodium, the amount of this substance varying with the diet. Where large amounts of food are taken and the metabolism is increased, the amount of calcium oxalate may be increased. In certain acid irritations in the genito-urinary tract, calcium oxalate may be increased in amount and the crystals deposited, sometimes forming calculi. They are particularly liable to form the nucleus of deposits of urates and phosphates.

The daily output of oxalic acid is normally about 10 mg., depending on the kind of food ingested, although a small amount of this acid may be excreted on an absolutely oxalate-free diet. When large amounts of this acid are excreted for some time, certain nervous symptoms are likely to be present, as occurs in neurasthenia and hypochondriasis.

Deposits of calcium oxalate may occur in the pelvis of the kidney and irritate the ureter and cause bleeding, as they are likely to be of irregular contour.

Oxalic acid may come directly from the food, may be produced from foodstuff in the body, or may occur in small amounts from some metabolism of the body tissues. While certain vegetable substances are probably most frequently the source of oxalic acid, such as rhubarb and spinach, it has been shown that oxalic acid may come from gastric fermentation of carbohydrates.[50] Proteins do not seem to cause the production of oxalic acid,[51] so that probably most of the calcium oxalate in urine comes more or less directly from preformed oxalic acid in the food. Consequently, the amount of calcium oxalate may be reduced to a minimum by a careful selection of the diet.

When calcium oxalate crystals are found in any amount in the urine, especially if the urine has not been standing long, preventive treatment of such deposits is essential. Although the danger of the formation of calculi is probably small, prevention by arranging the diet is so simple that this precaution should be taken. Besides the arrangement of the diet and the drinking of more pure water, the bowels should be regulated. The foods to be avoided, besides spinach and rhubarb, are cranberries, asparagus, radishes, horseradish, and many of the acid fruits, as grapes, currants and persimmons. Medicinal treatment should consist in the administration of acid sodium phosphate or some alkali, as sodium or potassium citrate.

URIC ACID

Uric acid is an end-product of nucleoprotein metabolism. The amount excreted in the urine varies from 0.4 to 1 gm. a day, and on a purin-free diet the normal individual excretes by the urine about the same amount of uric acid daily, that is, endogenous uric acid, or that which comes from systemic metabolism. Above 0.6 gm. of endogenous uric acid is considered a large amount. Between 1 and 2 per cent. of the total nitrogen of the urine comes from uric acid. In gout the uric acid may vary — it may be decreased, or, after an acute attack, it may be increased. A patient suffering from gout, given a known amount of purin-containing foods, will often excrete this exogenous uric acid more slowly than a normal patient; normally the whole of such exogenous uric acid is excreted in forty-eight hours. Patients with gout have shown more uric acid in the blood than normal persons, but this has also been found in other conditions. Uric acid in the blood is mostly in the form of monosodium urate. The amount of uric acid is increased in leukemia, whenever there is a breakdown of the white cells, when there is absorption of any blood or white cell exudate, and after active muscular exercise.

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[50] Baldwin: Jour. Exper. Med., 1900, v, 27.
[51] Wegrzynowski: Ztschr. f. physiol. Chem., 1913, lxxxiii, 112.

An increased amount of uric acid in the blood, so-called uricacidemia, is therefore found in most cases of gout, in severe nephritis, in leukemia, in resolving exudates such as pneumonia, in wasting diseases, and after Roentgen treatment, especially when the long bones or spleen are rayed.

Before it is decided that the uric acid output is increased, a twenty-four hour specimen of the urine should be examined, the patient continuing his regular diet and taking no drug. If it is found that the uric acid output is increased and there are uric acid and urate deposits, the treatment should be the same as that described for hyperacidity. The purins, such as meats, coffee and tea, should be removed from the diet, temporarily, at least.

The theory often advanced that an increased uric acid production is due to suboxidation is not tenable.[52]

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[52] Goodridge, F. G., and Foster, N. B.: The Relation of Uricolysis to Suboxidation, Arch. Int. Med., December, 1912, p. 585.

The retention of uric acid seems to be of more interest than an increased excretion. That at times the uric acid excretion is retarded and uric acid derivatives or by-products can be stored in the body in the joints seems unquestionable. That symptoms such as headache, indigestion, imperfect liver activity (perhaps an imperfect detoxication of toxins, or other absorbed products), constipation, myalgias, and joint pains may follow or accompany such retention seems to be a fact. The term "uricacidemia" should not be used too frequently, as uric acid is probably not frequently increased in the blood. An increased production and increased output of uric acid, or, in certain individuals, an increased production and retention of uric acid may at times be caused by drinking alcohol with a meat and purin meal, the alcohol causing a disturbance of the liver function and a greater mobilization of uric acid. Anything that removes this retained uric acid or limits the production will remove the symptoms described above.

Uric acid mistakes are not gout, and gout is not caused by uric acid malmetabolism. On the other hand, there is no question that in gout the uric acid metabolism is disturbed, that the metabolism of purins is often abnormal, that uric acid may be increased in amount, and that frequent attacks of uric acid retention occur in the general disturbance termed gout. Acute gout can occur without apparent retention of uric acid.

In uric acid retention there should be an elimination of meat purins, alcohol, and tea or coffee from the diet, and an increase in the amount of pure water consumed. Tobacco should be prohibited, or at least the amount used should be greatly diminished.

Salicylic acid will generally increase the uric acid excretion; alkalies may have the same effect. There is no good physiologic reason for administering lithium in any form, and the only value that lithium waters may have is due to the bulk of water drunk. Colchicum is not as valuable as a salicylate preparation, and its only value consists in a dosage sufficient to cause purging. Atophan (phenyl-quinolin-carboxylic acid) increases greatly the elimination of endogenous uric acid, both in persons suffering from gout and in normal persons. This increased elimination soon diminishes.

It has not been shown that uric acid causes a high blood pressure, although the intestinal disturbance accompanying uric acid retention may often allow resorption of intestinal substances which will raise the blood pressure. In gout also the blood pressure may be high, and there may be an increased suprarenal secretion. Some as yet unknown disturbance of internal secretion may be the cause of the apparently noninfectious chronic disturbance of metabolism which is termed gout. In the acute form of this disease, there may be nitrogen retention in the blood which will raise the blood pressure.

CHLORIDS

The daily excretion of sodium chlorid is from 10 to 15 gm., varying closely with the intake of salt. In health only a trace of chlorids is found in the stools, while varying amounts may be excreted by the skin, at least more than 1 gm. During starvation sodium chlorid is not eliminated, but is retained in the body.

The clinical point of interest in the chlorids is their retention by the body and the part this retention plays in edema. This point will be discussed fully later under the heading of edema. In pneumonia, and in some cases of profuse catarrhal exudate, the urinary excretion of chlorids is diminished without retention, owing to its excretion elsewhere.

INDICANURIA

Indicanuria is of frequent occurrence, and indicates that some protein putrefactive process is taking place in the ileum and colon with the production and absorption of indol and the excretion of indoxyl potassium sulphate (indican). Indol and skatol are derived from bacterial putrefaction of tryptophan, an amino acid. They are absorbed from the intestines, becoming oxidized. The indoxyl reaches the liver and combines with sulphuric acid, forming indican, which finally passes off by the urine. On an ordinary diet from 5 to 20 mg. of indican are daily excreted in the urine. On a diet very low in nitrogen there may be practically no indican eliminated.

Indican in the blood is not poisonous, but other products of decomposition and toxins absorbed from the intestines at the same time as the indol may produce symptoms of poisoning. If indol alone does not, skatol and cresol with toxalbumin will frequently produce symptoms of poisoning, such as headache, restlessness, insomnia, gastro-intestinal indigestion, at one time a dry skin, at another time more or less profuse perspiration, sometimes eruption on the skin, and sooner or later kidney irritation, with attacks of polyuria, traces of albumin, cylindroids, and even hyaline casts.

Not every case of putrefaction in the intestines causes indicanuria, nor does constipation per se cause it. It occurs acutely in abdominal disturbances, as typhoid fever, in some types of diarrhea, in intestinal obstipation, and in peritoneal inflammations. Indicanuria is only an indicator of a condition, namely, putrefaction in the lower part of the intestine, and shows that other irritant and poisonous products are being absorbed. If this absorption persists, as shown by persistent indicanuria, these products of protein decomposition irritate the walls of the blood vessels, or stimulate the suprarenals to greater activity in producing vasopressor substance; or both conditions prevail, resulting in hypertension. If the liver and kidneys are equal to the strain, no evident symptoms of ill health may result for months or years; but the persisting hypertension and continued irritation with perhaps other factors, finally causes cardiovascular-renal disease.

Other symptoms caused by such fermentative processes in the intestine are: irregular bowel action (constipation or diarrhea); gastric flatulence; coated tongue; dizziness; drowsiness during the day and insomnia or troubled sleep at night; irritability; at times muscular lameness or muscle cramps; cold extremities, often accompanied by increased perspiration due to dilatation or contraction of the local blood vessels caused by irritation; polyuria if there is hypertension, and a slight yellowing of the sclerotics and some parts of the body, as the palms of the hands if there is liver disturbance, the perspiration becoming malodorous and staining the clothing. These symptoms are often termed "biliousness," and fortunately the treatment generally given with that diagnosis is successful, namely, calomel or some other purgative, together with a more rational diet, including the reduction of the amount of meat, alcohol, coffee, etc., consumed.

In addition to intestinal and reflex causes, and intestinal putrefaction caused by improper food or a too large amount of meat proteins, probably other frequent causes of putrefaction are pus, abscesses, etc., in the mouth, throat or nasal sinuses. Bacteria swallowed with the food from pyorrhea alveolaris, and bacteria or toxins absorbed into the blood from infection at the apex of a tooth doubtless play a large part in producing fermentation in the small and large intestine. It should be noted that authorities on medicine have long stated that pyorrhea alveolaris and chronic colon infection or putrefactive processes are of common occurrence after 40 years of age.

As there is usually a small amount of indican in the urine of a normal person, no examination of urine is complete unless it is noted whether or not indican is increased. If it is found that the amount of indican is increased, there may be a disturbance of the lower part of the small intestine and the colon. Animal proteins should be absolutely removed from the diet for a time, and there should be thorough purging. The cause of the disturbance should be sought and removed, if possible. This means particularly careful examination of the teeth, gums, tonsils, nose and adjacent sinuses. If no tangible cause for indigestion can thus be found, it must be considered a functional condition such as has just been said to be of common occurrence after the age of 40. However, we have not yet reached the stage of postponing old age by removing the colon. Neither can we prolong youth or life by injecting lactic, Bulgarian or other bacilli or by taking sour milk or yeast at frequent intervals. Although an attempt to overcome colon fermentation should always be made, the seriousness of this condition should not be exaggerated, as it is a condition well tolerated.

The condition of the patient having been ascertained, the bowels should be caused to move daily and regularly; a more or less rigid meat purin-free diet should be ordered, and colon washings may be given until the urine is practically indican-free. The flora in the intestine may be changed by administering yeast or lactic bacilli; but it should not be expected that such treatment alone can cure a colon putrefaction. These bacilli should not be given over too long a period, as they may predispose toward joint pains and symptoms of a general hyperacidity. Liquid petrolatum is now much used, and often seems beneficial, especially if the patient is subject to constipation. When it is administered over too long a period it frequently causes gastric and intestinal indigestion, perhaps by mechanically interfering with the secretions.

Many persons have intestinal disturbances because they become chilled at night, the blood being driven from the surface of the body to the abdomen. These intestinal disturbances, colon putrefactive processes, and the formation of large amounts of colon gas are often prevented by keeping the body, and particularly the bowels, warm. Phenyl salicylate (salol) in a dose of 0.25 or 0.30 gm., in capsule, three times a day, after meals, for a short period is often of benefit in preventing intestinal fermentation.

The patient may be given one meat protein after another to see which he tolerates without decomposition. He should learn, however, so to regulate his diet and bowels as to prevent colon fermentation. It is well to set aside one day each week on which to abstain from protein food, and to take a saline purge on that day (if such is not contraindicated by the condition). A sour milk week occasionally is of value.

Care must be taken that anemia is not caused by a limited diet. Anemia, however, may develop from persistent putrefaction. The weight should be watched and the skin studied for eruptions, dryness or profuse perspiration, in order to regulate properly food, drink, and possible drugs. Hypothyroidism or hyperthyroidism may be the cause of the intestinal condition.

This discussion, though apparently a long one on a simple condition, is a discussion on preparedness for the proper management of a patient with intestinal indigestion; and indicanuria may mean prevention of kidney, cardiac and vascular disease. It should be noted that epilepsy and some mental disturbances have been cured by removal of bowel defects. Intestinal stasis can also aid bacterial invasion, particularly colon bacilli disturbances, and many other more or less serious ailments. As first stated, a tangible cause for putrefaction and indicanuria must be sought through any or all of the gastro-intestinal examinations now so well understood, from gastric and fecal analyses to bismuth pictures. Chronic appendicitis, chronic gall-bladder trouble, and anything that damages or irritates an intestine may cause intestinal disturbance and absorption of toxalbumins and the substances already mentioned. Passive congestion of the liver and intestines from an insufficient heart is a prominent cause of disturbances resulting in indicanuria.

It has been stated that whenever there is nitrogen retention in the blood there is indicanuria.[53]

INDOLACETIC ACID

This substance, probably a derivative of tryptophan, has been found in urine, and Ross[54] thinks that it must be due either to a constitutional disorder of metabolism or to a particular kind of intestinal putrefaction.

FAT (LIPURIA)

A minute amount of fat (0.02 gm.) is excreted daily. This amount may be increased to 34 mg. by eating large amounts of fat, according to Sakaguchi.[55] He investigated the urinary fat output in various chronic diseases, and found no disturbance of this minute urinary excretion. The fat of the urine may, however, be increased in chronic nephritis when there is fatty degeneration of the kidneys, and is probably usually increased in phosphorus poisoning.

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[53] Tschertkoff, J.: Deutsch. med. Wchnschr., 1914, xl, No. 36.

[54] Ross, E. L.: A Preliminary Note on the Excretion of Indolacetic Acid in the Urine, Arch. Int. Med., July, 1913, p. 112.

[55] Sakaguchi: Biochem. Ztschr., 1913, **48**, 1.

CHYLURIA

This is a rare condition in which a milky urine is due to chyle entering the urinary canals directly from the thoracic duct. Such a condition is generally caused by the burrowing of parasites called filariae. An unexplained chyluria has rarely occurred.

CYSTINURIA

Cystin rarely appears in the urine. Its appearance is due to improper metabolism in the body. Cystin is an amino acid derived from protein, and is normally transformed in the body, its sulphur appearing in the urine as a sulphate. Infrequently cystinuria causes deposits in the pelvis of the kidney or in the bladder. This condition should be treated by a reduction of the amount of protein consumed and by the administration of alkalies. Cystin may be produced by the body proteins, and an endogenous cystinuria may sometimes occur.

ALBUMOSURIA

Excretion of proteoses in the urine constitutes the condition called albumosuria. It may occur in suppurative conditions or processes such as that which takes place during the resolution of a serious pneumonia in which a considerable portion of lung has been involved. It has occasionally been found in leukemia.

BENCE-JONES PROTEIN

The abnormal protein substance termed Bence-Jones protein is probably of endogenous origin. It is generally combined with anemia and does not seem to be a decided symptom of anything, but multiple myeloma or some form of bone-marrow disease. The prognosis for this condition is unfavorable.

UROBILINURIA

Bilirubin is converted in the intestines by bacterial action into urobilinogen and urobilin, which are absorbed by the blood and converted into bilirubin in the liver. Traces are usually found in the urine. When an increased amount appears in the urine, the condition is termed urobilinuria.

BILIRUBINURIA

Any obstruction of the biliary ducts will cause absorption of bilirubin into the blood, resulting in jaundice, and bilirubinuria. The urine becomes brown, and when shaken shows a yellow froth.

If bilirubin appears in an increased amount in the urine it denotes a more or less serious liver disturbance. If a small amount of this bile substance passes off in the urine it is not harmful to the kidneys; but if a large amount is excreted during an extended period of time, renal irritations, possibly accompanied by albuminuria, may occur.

ALBUMINURIA

Albuminuria may be classed as follows:

1. A symptom of nephritis.
2. Accidental albuminuria causes:
    - (*a*) Dietary (alimentary).
    - (*b*) Chilling of the body.
    - (*c*) Unexplained (frequently due to focal infection).
3. Incidental albuminuria causes:
    - (*a*) Cold baths.
    - (*b*) Menstruation.
    - (*c*) Athletics or other physical strain.
    - (*d*) Cardiac weakness.
    - (*e*) Irritation in some part of the urinary tract.
    - (*f*) Hypertension.
    - (*g*) Ether or other anesthesia.
4. Orthostatic (lordotic, cyclic, adolescent).

The term "albuminuria" refers to the presence of serum-albumin in the urine. Nucleo-albumin may be present occasionally in a patient who is in perfect health. Frequently nucleo-albumin and serum-albumin are both present, particularly in acute or subacute nephritis. Nucleo-albumin is rarely present in chronic nephritis. The presence of this substance alone does not ordinarily mean that there is a kidney lesion. Albumin in the urine is either excreted by the kidneys or comes from some part of the urinary passages. If it results from some localized irritation, inflammation or hemorrhage, there are more white cells present in the urinary sediment than in ordinary albuminuria. If there is hemorrhage, there are, of course, red cells. A small amount of albumin is found in cystitis, and a somewhat larger amount in pyelitis. The amount, however, is always small unless it comes directly from the kidney secreting substance. Urine containing a small quantity of sodium chlorid or other salts may show just a trace of serum-albumin, but if sodium chlorid is added to such urine, a larger deposit will be obtained. Frequently urine in which albumin has previously been found becomes free from this substance because the patient is passing a larger total amount of urine; in other words, if he has been given a diuretic or a large amount of water, albumin may not be found, while the same condition which caused the albuminuria still exists. Circulatory tonics, such as digitalis, provided the blood pressure is not high, frequently, temporarily at least, may cure an albuminuria.

1. The albuminuria of nephritis will be discussed later under that heading.

2. Accidental albuminuria may occur from several causes. It is sometimes due to exposure to cold; more frequently it is due to some dietary error, such as overloading the system with proteins; or it may be due to some alimentary disturbance. Traces of albumin are often found in the urine in intestinal disturbances, particularly in chronic diarrheal conditions. Albumin is often found in the urine without any assignable cause, probably owing to some focal infection. Mouth and throat infections may cause serious kidney disease; mild irritations, therefore, may cause the appearance of small amounts of albumin in the urine.

3. Incidental albuminuria is frequent. It may be caused by cold baths or cold showers. It may occur with menstruation or when menstruation is delayed. It occurs frequently after severe muscle strain, as in competitive athletics.

Incidental albuminuria as a symptom of an acute disease, and its appearance during the intensity of an acute infection have been proved. Backache and the primary symptoms of most acute infections are probably due to kidney congestion.

Cardiac weakness or insufficiency, low blood pressure, or anything which causes congestion of one or both kidneys may allow leakage of albumin. On the other hand, a small trace of albumin is frequently a symptom of hypertension, and may be the forerunner of actual kidney disease.

Albumin frequently appears in the urine following an operation, and is perhaps chiefly due to the anesthetic. It may, however, be due to some position in which the patient is compelled to sustain himself for an extended period of time, such as the lithotomy position; or sluggishness in the circulation of the kidneys may occur from prolonged lying on the back.

Renal irritations, or irritations in some part of the urinary tract, frequently cause the discharge of small amounts of albumin in the urine. A floating kidney may also be the cause of this condition.

When the cause of albuminuria has been determined, the preventive treatment is self-evident. If it is a part of some diseased condition, it will diminish with any improvement in that condition.

4. Orthostatic albuminuria (lordotic) has also been called the albuminuria of adolescence, because it is apt to occur in developing children. As it occurs only when the individual is active, it was for many years supposed to be entirely due to a change in the blood pressure of the kidney vessels, and was termed orthostatic albuminuria, as in the majority of instances the albumin disappeared from the urine if the child remained in bed. Occasionally the administration of cardiac tonics and vasopressor drugs improved or prevented the condition. This symptom disappeared in many of the children as they grew older, the duration

of the condition varying. However, since Jehle published his monograph, in 1909, stating that so-called orthostatic and functional albuminuria was often due to lordosis causing passive hyperemia of the kidneys, the truth of his observations has been acknowledged by many investigators. It seems probable that some inherent weakness of the kidney circulation must also be a factor, though general vasomotor disturbances and low blood pressure have not been constantly present. Not every lordotic person is afflicted with albuminuria; nor is every orthostatic albuminuric lordotic. However, lordosis is apparently a frequent cause of albumin in the urine in an active patient.

Albuminuria due to lordosis may come from only one kidney, as has often been proved by ureteral catheterization. In schoolchildren it may be the result of sitting for some time daily with the hands folded behind the back.[56] Albuminuria may also be caused by a movable kidney, due to venous or passive congestion.

The term "orthostatic albuminuria" should be limited to that albuminuria without casts which occurs after standing or moving about, and which disappears with a reclining position. If casts and blood cells are associated with this condition, the patient may be suffering with nephritis. Hence position may be the final cause of sufficient passive congestion for the excretion of albumin, casts and blood cells. The necessity for complete rest, in even slight kidney congestion, is therefore obvious.

Orthostatic albuminuria may be due not only to lordosis, which produces a passive congestion of otherwise normal kidneys, but also to an insufficiency of the kidney circulation, possibly congenital, with or without lordosis. This condition may also be caused by kidneys of insufficient size for the individual. Orthostatic albuminuria may also be caused by certain positions or labors which cause a profusion of blood to course to the kidneys, resulting in passive congestion and leakage of albumin. Sometimes in rapidly growing youths the kidneys may be insufficient, just as we find hearts insufficient. Later the kidneys grow to the body size.

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[56] Piesen: Wien. klin. Wchnschr., Jan. 5, 1911.

Lordotic albuminuria or albuminuria due to disturbed circulation in otherwise normal kidneys usually occurs between the ages of 10 and 15. Scoliosis may also be a factor in causing albuminuria. Tall, thin children with poor circulation are most frequently affected. This insufficiency of circulation is evidenced by cold hands and feet, and general weariness after exercise. Functional or orthostatic albuminuria, or albuminuria occurring after hard exercise, may be due to a kidney which has imperfectly recovered from a previous inflammation. This kidney may be insufficient for years and become a predisposing cause of chronic nephritis.

The prognosis of this albuminuria of adolescence is generally favorable, although it usually shows a lack of kidney tone or imperfect function, and may indicate that the surrounding circulation is not perfect. Hence there is danger of serious disturbance later, particularly with any added strain on the kidneys.

In intermittent albuminuria, although casts may be absent, there is always the possibility of development into a condition of nephritis; consequently insurance companies look on these cases with suspicion. However, patients with intermittent, lordotic or orthostatic albuminuria not infrequently recover completely and permanently.

Before an albuminuria is considered a simple albumin leak, all suspicion of more serious conditions of the kidneys must be eliminated by examinations of the urine under varying conditions. Casts must be absent, and there must be no cardiac hypertrophy or other symptoms which would show that the kidneys are suffering from localized or general inflammation. If granular casts are found, the prognosis is not good, but may not be very bad. Determination of the absence of any focal infection, tests of function, blood examinations, and, if advisable, the passage of ureteral catheters to show whether both or only one kidney is at fault will make the prognosis clear.

The results of urine examined on first rising in the morning, and again in the latter part of the afternoon will quickly show whether the condition is an orthostatic albuminuria. Also, tests of diet and exercise will show the limitations of the kidneys and their ability to sustain effort without leaking albumin.

Temperature tests should be made, as it is not unusual to find that so-called functional or orthostatic albuminuria is due to the fact that a tuberculous kidney is developing. Such a condition may be discovered before pus or blood appears in the urine. The localization of an albumin leak in one kidney, with some rise of temperature in the evening, should cause not only the centrifuged sedimented urine to be examined for tubercle bacilli, but also the guinea-pig test to be made.

The spine should of course be examined for scoliosis and lordosis, and proper orthopedic treatment should be instituted if either of these conditions is found. Also, any faulty position assumed by the child while studying in school or at home, or such as results from lying in a hammock or sleeping in a bed with too weak springs, should be corrected.

Disturbed menstrual function in young girls is at times a factor in causing leakage of albumin. When normal menstruation occurs spontaneously or as the result of appropriate treatment, such albuminuria ceases.

That hard physical exercise may cause albumin and casts to appear in the urine is now too well known to require proof or discussion. That a repetition of this condition in athletes may cause no future harm will not be so readily accepted, though Jundell and Fries,[57] after repeatedly examining the urine of 163 athletes after exercise of various kinds, concluded that the kidneys are never permanently injured. They did not, however, take into consideration the more or less permanently increased blood pressure caused by such exercise. It would seem only logical to believe that albuminuria caused by athletics represents kidney tire or kidney strain, making these organs more susceptible to irritation, and must be as harmful as overindulgence in improper food, frequent diarrhea, repeated attacks of rapid heart from coffee or other cause, periods of insomnia, etc.

In the treatment of such recurrent albuminuria or in the treatment of lordotic albuminuria, strenuous exercise should be forbidden, and all functions of the body should be made as perfect as possible. The diet should be properly arranged; a great deal of rest taken; muscle exercises to increase the strength of the back muscles advised; anemia treated by the administration of iron; a weak heart aided by cardiac tonics; constipation relieved; plenty of fresh air prescribed; and sea bathing and cold bathing usually prohibited—every means being used to improve the general condition of the patient.

Of course it should be recognized that traces of albumin may be caused by renal concretions. A growing child or youth with traces of albumin in the urine, and with low blood pressure may develop pulmonary tuberculosis.

ALBUMINURIA IN INSURANCE

A broader point of view and better knowledge of functional or accidental albuminuria have made its discovery in applicants for insurance a cause for reexamination of the urine, and not a cause per se for rejection. That albuminuria is not normal is acknowledged, and when it is found more than once in an applicant he is either rejected or his acceptance postponed. The age of the person in whom it is found is of importance, as after 40 even traces of albumin are suspicious. For the same reason an occasional hyaline cast may not be alarming in persons under 30; in those over 40 it is ominous. An applicant in whom albumin is found should be reexamined unless the amount is considerable. After four negative specimens and an absence of casts, with a blood pressure below 140 mm. systolic, or with a diastolic below 100 mm. if the systolic is 150, with cardiac sufficiency, and with no history of syphilis, tuberculosis or any renal disease, the applicant should be accepted. Any signs of cardiac hypertrophy or symptoms of hypertension should cause the applicant to be rejected.

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[57] Jundell and Fries: Nord. med. Ark., 1911, **44**, Internal Medicine, No. 1.

A specific gravity of anything less than the whole twenty-four hours' urine is unimportant. A single specimen may vary from 1.005 (nervous urine, or urine passed soon after a long summer drink, or in severe cold weather) to 1.025, and still not indicate the average specific gravity. A high specific gravity, namely, more than 1.025, without sugar and without normal sediment, is rarely found in applicants for insurance. An abnormally low specific gravity of the urine is frequent, and in such cases most insurance companies require another examination; such an examination, however, is not justified on these data alone.

As the action of tobacco on the heart, blood vessels and kidneys is positive, a question of great importance, which the applicant should answer, is, "How many cigars, cigarets, and pipes do you smoke a day?" This is not yet often asked on insurance examinations, but is just as important today (there being fewer men who drink to excess) as is the question, "How many glasses of beer or whisky do you drink a day?" It is not sufficient to determine that the blood pressure is not too high in those who smoke to excess, as the high blood pressure may have become normal as a result of the weakening of the heart muscle by tobacco.

Occasionally a peculiar nervous albuminuria occurs in certain persons while under some nervous tension. However, although this condition often accompanies such nervous tension, these people never give evidence of orthostatic albuminuria. It is, of course, possible that at times some associated but undiscovered cause is present.

There are also instances of albuminuria in active persons with casts in the urine for years without any apparent progression, whose death will not be due to cardiovascular-renal disease. In such cases data of kidney function tests and of the nonprotein nitrogen of the blood should be obtained. We can no longer assume that hyaline or a few granular casts demonstrate nephritis. They do demonstrate some abnormal congestion or disturbance in some part of the kidney, but such a disturbance may completely disappear, as can a mild disturbance in any other part or organ of the body. On the other hand, coarse granular, fatty and waxy casts show kidney inflammation.

Traces of albumin may result from inflammation of the prostate and of the seminal vessels, and, of course, from pus anywhere in the urinary tract. In albuminuria from vesiculitis, Belfield[58] finds that if the urine is passed into three glases, a little into the first, the bulk into the second, and a little into the third, a good reaction is found for albumin in the first and last glasses, but less in the second glass; while in renal albuminuria all of the urine tests the same. In prostatitis, massage of the prostate will increase the albumin in the urine passed immediately afterward. This is especially significant if the bladder has been previously washed. Of course urine drawn by ureteral catheters will positively prove the source of the albumin.

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[58] Belfield, W. T.: Vesicular Albuminuria, The Journal A. M. A., June 19, 1915, p. 2040.

CASTS

There are several kinds of tube casts: (1) hyaline; (2) epithelial; (3) blood; (4) granular; (5) fatty; (6) waxy or amyloid, and rarely (7) bacterial. Cylindroids formed from so-called mucus are shorter and broader than hyaline casts, but are less firm in structure, being readily broken up. They are of no significance except when they occur in considerable numbers, when they indicate kidney irritation and an abnormal increase of mucous secretion. Cylindroids and hyaline casts are formed from the same substance as that which forms the mucous threads sometimes found in the urine.

1. A hyaline cast is a secretion of this mucus, perhaps by the glomerulus, which has remained in a tubule until it has become formed and shaped, although its size may not represent the region of the kidney from which it comes, as the tube may have become dilated. Most hyaline casts probably come from the Henle loops. Albumin passing through the glomeruli may also coagulate and form hyaline casts. It is probable that a hyaline degeneration may occur in certain pathologic conditions and in acute infection of the epithelial cells of the tubules, and hyaline casts be thus formed. Both cylindroids and hyaline casts are more likely to be found in concentrated and acid urines, as low specific gravity urine and neutral and alkaline urines cause disintegration of these frail structures.

Like cylindroids, hyaline casts indicate only kidney irritation and a sluggish urinary flow in some part or parts of the kidneys. Of course the freer the flow of urine, the shorter time will mucus or any other substance remain in the tubule.

High power microscopic examination shows hyaline casts to have many organized particles and not to be absolutely homogeneous.

Not only hyaline, but also epithelial and fine granular casts may be found in the centrifuged urine of normal persons, especially if this urine is first sedimented. However, they usually represent some slight kidney irritation. If hyaline casts are repeatedly found in the urine of the same person, particularly if he is over 40 years of age, and if there are traces of albumin, it may indicate the beginning of chronic interstitial nephritis; but it should be emphasized that hyaline casts can occur with physical strain, with mental anxiety and mental strains that would disturb general metabolism or the circulation, and with any acute infection, even the simplest.

2. Epithelial casts represent hyaline casts with loosened epithelium of the tubules, and the form of the epithelium may denote the region of the kidney in which the casts originated. Irritation or passive congestion may cause this type of casts to occur in slight disturbances, as well as in tubular nephritis.

3. Blood casts show acute kidney congestion or kidney hemorrhage, and are composed of blood cells and serum. Many epithelial, hyaline and finely granular casts may show both white and red corpuscles. Blood casts are most frequent in acute nephritis. When found in hematuria, they show that the bleeding, in part at least, is from the kidney structure. Even when in acute nephritis the blood casts are in large amount, they may not be of serious omen unless the patient is

uremic. With signs of uremia and serious kidney congestion, the prognosis is unfavorable.

All urines will contain some leukocytes and frequently red cells, which may come from any part of the tract. However, unless the urine has been obtained by the ureteral catheter (without injury), or there are blood casts or blood cells in casts, it cannot be positively stated that these blood cells come from the kidney.

4. Granular casts are degenerated epithelial casts. When there is a diffuse nephritis and degeneration occurs in the epithelial cells of the convoluted tubules, more or less débris is thrown into the lumen of the tubules which, remaining there for some time, causes the production of granular casts, in the beginning, with fine and later with coarse granulations. When, with degenerative disease of the epithelium of the tubules, there is a large amount of urine passed, the débris may not remain in the tubules long enough to form casts; consequently such urine, when centrifuged, will show epithelial and granular débris. Also, long and rapidly centrifuged urines may have the granular casts broken down, forming débris.

5. Fatty or oily casts represent fatty degeneration of epithelial casts. They may be composed wholly of fat or of oil, or may be epithelial or granular casts containing oil globules due either to an infiltration or to degeneration of the cell contents. They generally indicate a serious diseased condition, and occur when there is chronic nephritis of any type which has been of considerable duration.

6. Waxy or amyloid casts are somewhat like hyaline casts, but show amyloid reaction. They are not necessarily confined to amyloid infiltration or degeneration of the kidneys, but are signs of severe nephritis, and may be due to a degenerated blood coagulum. They

are either straight, bent or twisted, and represent molds of the convoluted tubules. The hemorrhage may be from the glomeruli, the blood remaining in the tubules, or it may come directly from the walls of the tubules. It should perhaps be emphasized that waxy casts do not necessarily denote amyloid disease of the kidneys.

7. Serious kidney infection is sometimes accompanied by casts of bacteria in the urine. However, before deciding that these casts come from the kidneys, one must be certain that the urine examined is fresh, as rolls of bacteria resembling casts frequently occur in old urine.

Casts may come from one or more infected regions of the kidneys, but may represent an acute or chronic disturbance of just one portion. They do not disturb the general function of the kidneys, though they must impair that of the parts affected. In this relation we should bear in mind the great recuperative power of the kidneys.

RENAL CALCULI

Renal calculi vary from small particles of precipitated salts—enough to make a granular sediment in the urine, passed without pain or knowledge, to concretions large enough to cause colics, or to remain in the pelvis of the kidney. Although it was thought that uric acid stones were very frequently found in the urine and that calcium oxalate stones seldom appeared, it has been shown recently that the latter occur most frequently, and form the largest element in the composition of renal calculi. It has been shown also that these calcium oxalate calculi are not necessarily hard.[59] As uric acid calculi may be very hard, and calcium oxalate calculi may be soft, Rowlands believes that the hardness of calculi depends on the rapidity of their formation — the slower the formation the harder the calculus.

Most renal calculi contain uric acid or urates in small amounts. However, these ingredients can be determined only by chemical analysis, as one cannot decide the character of a calculus from its color, shape and hardness. Renal calculi may occur with or without gout, that is, gout and the formation of calculi are not closely related, according to Kahn. The tophi in gout may be composed of sodium and ammonium biurate or possibly of calcium oxalate. It is by no means to be taken for granted that they always contain uric acid or urates.

Phosphatic calculi frequently have a uric acid nucleus. Theoretically calcium oxalate and uric acid calculi should not be redissolved in the kidney or bladder after having once been formed; nevertheless, urate and phosphate deposits evidently do dissolve or break down repeatedly. However, it has recently been shown by Haskins[60] that all neutral, alkaline or slightly acid urines will absorb uric acid; that the less acid the urine, the more uric acid will generally be dissolved, and that a dilute urine will usually dissolve more uric acid than a concentrated urine. Therefore, a freshly formed uric acid concretion may dissolve in the kidney or bladder, provided the urine is not too acid or too concentrated.

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[59] Rowlands: Biochem. Jour., 1908, **3**, 346. Mackarill, Moore and Thomas: Biochem. Jour., 1910, **5**, 161. Kahn, Max: Study of the Chemistry of Renal Calculi, Arch. Int. Med., January, 1913, p. 92.

The phosphate calculi are usually mixtures of ammoniomagnesium phosphate and calcium phosphate. Calcium carbonate is rarely found; xanthin and cystin deposits occasionally occur. Some irritation in the pelvis of the kidney is generally the primary cause of this deposit, a mucous shred, or a clump of bacteria with its surrounding leukocytes forming a nucleus.

SYMPTOMS

While patients can pass a large amount of gravel with little pain, the presence of these crystals in the urine is frequently accompanied by backache or pains shooting down the ureters, irritability of the bladder, and occasionally urethral stinging from the urine. If a calculus is present in the pelvis of the kidney, repeated examinations of the urine and of the centrifuged portions will usually prove that there are more red blood corpuscles than normal. The urine may show macroscopic blood and shreds of mucus streaked with blood. Of course, blood in the urine does not prove that a calculus is the cause; but with other symptoms of calculi, with pain referred to one side, with ureteral catheterization showing blood coming from one kidney, and with the positive evidence of a roentgenogram, the diagnosis can be made. It should, of course, be recognized that kidney pain and even kidney colic may not be caused by a calculus impinged in the orifice of the ureter. The cause of this usually intermittent pain may be discovered by deduction or by the examinations just suggested.

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[60] Haskins: Jour. Biol. Chem., August, 1916.

Sometimes this kidney pain is not referred to the back, but to the center of the abdomen, upward toward the liver, to the right side, or to the appendix region. If the calculus has entered the ureter, the pain is referred down its length toward the bladder; soon pain is felt in the testicles in the male, and in the vulva in the female, and later, in the male, there is pain referred to the end of the penis. It should be noted that renal calculi occur more frequently in men than in women.

Besides blood, albumin may occur in the urine, and hyaline casts may be found, owing to reflex irritation caused by the calculus. If the pelvic irritation continues, inflammation may result with increased mucous secretion, followed by pus.

Generally careful palpation of the suspected kidney will show that it is tender to pressure; it may be enlarged.

A true renal colic comes on suddenly, often without previous symptoms, and is as likely to occur when the patient is in bed as when he is undergoing physical exertion. In about half of the cases, pain starts in the lumbar region of the affected side, comes gradually around toward the abdomen, and shoots down toward the bladder, as described. The pain is generally accompanied by nausea and vomiting, a feeling of faintness, and a weak pulse. Sometimes the skin is covered with cold, clammy perspiration, and the patient is on the verge of collapse. At times the patient lies down with his pain; sometimes he is bent over with pain, and sometimes he writhes in agony. There is often a desire to urinate frequently, sometimes without the ability to do so. The temperature seldom rises. Blood may pass into the bladder, the urine becoming discolored even before the calculus has passed. When the calculus reaches the bladder, the cessation of pain is almost instantaneous. During its passage down the ureter the patient is generally aware of its location, the pain leaving the back and being referred more and more toward the bladder and down into the genitals.

As soon as it has been determined that the calculus has reached the bladder, the patient is told to hold the urine for a while and then pass it as rapidly as possible from the urethra, so as to flood out the calculus from the bladder. Until the calculus is found, the urine should be passed either into a jar or through a strainer, and neither physician nor patient should be content until the calculus is found, or until it has been decided that the calculus has disintegrated somewhere during its passage. Such a disintegration often occurs if the calculus has been rapidly formed and is soft, especially if it is of the urate variety.

Recurrent attacks of renal pain, and often apparently ureteral pain, may occur without the passage of a calculus. Such attacks are often accompanied by fever, and may be preceded by a chill. If the pain is not very severe, it may be termed a backache, and the patient be told that he has malarial intermittent fever. It is inexcusable, however, for such a diagnosis to be made, as the malarial element can be excluded by examination of the blood; and a careful examination of the urine, localization of the symptoms and roentgenoscopy should make the diagnosis conclusive. In these instances not only does a large amount of mucus appear in the urine, but soon small amounts of pus are apt to appear, with signs and symptoms later of actual inflammation of the pelvis of the kidney.

TREATMENT

The immediate treatment is the same as that of colic of the kidney. The pain must be stopped and the tissues relaxed if possible. No manipulations of the kidney or urethra should be attempted, as this will do more harm than good. The patient should receive a hypodermic injection of ¼ grain of morphin with 1/100 grain of atropin, and, if possible, should sit in a bathtub filled with warm water up to his waist. The relaxation caused by the water may stop spasm and aid the passage of the calculus. Of course if the pain is intense and he is faint and vomits, or if the pain persists for an extended period of time, such bath treatment is impossible and inadvisable. Under such conditions, hot, moist fomentations over the affected side will afford some relief.

If the morphin has no effect within from a half to three quarters of an hour, another injection must be given, the character of the pain and the size of the person determining the dosage. Another ¼ grain or ⅛ grain of morphin should be given, not combined with atropin, as it is unwise to repeat the dose of atropin. Atropin acts slowly and persistently, and a large dose may cause stimulation of the heart and central nervous system instead of sedation of the peripheral nerves. If the patient has been seen previously and is known to be very tolerant to morphin, the first dose may be much larger than the ¼ grain advised; large doses of morphin should not be given, however, since as soon as the calculus is either forced back into the pelvis of the kidney, or passes into the bladder, the intense pain immediately ceases, and the whole activity of the morphin is in evidence. In fact, whenever large doses of morphin have been administered for acute pain and the pain has ceased, the patient should not be allowed to sleep for some hours, the nurse or attendant keeping him awake until part of the morphin has been eliminated. The patient should be encouraged to pass the urine as soon as the bladder is considered full, and large drafts of water should be taken to wash out the stomach. Black coffee may also be given to stimulate the nervous system and to counteract the effect of the morphin on the respiratory center, and later, the patient should be given a cathartic to relieve constipation caused by the morphin. Food should not be given for some hours.

In order to avoid an overdose of morphin, the patient may be given a few whiffs of chloroform to bring about the relaxation desired and to allow the morphin to show its effect. Sometimes the action of chloroform administered after a morphin injection is successful, the pain not recurring with such intensity, and the patient becoming quiet, although he knows that the stone has not passed. Much chloroform should not be given, nor should it be given too frequently, as cardiac debility may be caused by the repeated use of even small inhalations. No other drugs are of any assistance whatever in the treatment of renal colic.

After-Treatment.—The calculus should be examined in order to discover its component parts, as its constituency may indicate the character of the food to be avoided. It should be remembered that at the time of the deposit of one calculus there may have been others, and prophylactic measures should be taken, as other colics may occur. The patient should drink large amounts of distilled water, or, if the urine shows high acidity, particularly if the calculus was found to be of the uric acid variety, he should be given alkalies and large amounts of sodium bicarbonate or good sized doses of potassium citrate. The frequency and size of the dose depends on how much it takes to cause the urine to become alkaline. On the other hand, if the calculus was found to be phosphatic and the urine only faintly acid, or alkaline, alkalies should not be given, but rather something to make the urine acid — as hexamethylenamin or dilute hydrochloric acid — to be taken after meals.

The diet depends on the character of the concretion and the condition of the urine. Meat should for a time be avoided, provided the urine is highly acid and the calculus was composed of uric acid, calcium oxalate or urates. If the urine is alkaline and the calculus was composed of alkaline salts, meat should be allowed. Ordinarily, a milk and cereal diet is proper, the diet depending more or less on the amount of intestinal indigestion caused. Such indigestion is to be guarded against both by the administration of laxatives and by arrangement of the diet, as the formation and absorption of by-products or toxins, and the presence of indicanuria have a tendency to cause more urinary deposits. Needless to say, alcohol, excessive tea and coffee drinking, rich foods and fancy dishes should be prohibited. Pure water is of great importance, and the patient should drink it liberally unless some affection of the heart or kidneys compels him to drink it in moderate quantities. There is no artificial water, no mineral water, no lithia water which will dissolve as much salts as will distilled or ordinary pure water. Consequently, nothing is attained by buying expensive bottled waters.

In the presence of the conditions that cause urinary concretions, it is self-evident that a sojourn at some mineral spring, "cure" or sanatorium at which the diet, the exercise, and the hours of sleep and rest are all under supervision, will benefit such patients; but let it be emphasized that it has not been proved that lithia waters will dissolve renal or gallbladder calculi, while lithia in almost any form, in any tangible amount, tends to cause gastro-intestinal indigestion.

The subject of urinary solvents has recently been reviewed by Haskins,[61] whose findings may be thus summarized: Piperazin may cause the urine to dissolve more uric acid, which may be further increased by giving with it sodium citrate or bicarbonate. The best results occur when diuresis is not caused. Lysidin increases the uric acid solution, but is not a very good agent, as large doses are required. Lithium carbonate is of value only in large and dangerous doses, and is no better than sodium citrate or bicarbonate; in fact, sodium citrate, sodium bicarbonate or potassium citrate are very satisfactory uric acid solvents when given in sufficient doses to alkalize the urine. In other words, there is no necessity for administering any but these last three well known, simple drugs, especially when combined with the use of distilled or pure water.

Unless it is desired to increase the uric acid excretion it is not advisable to administer atophan or novatophan in cases with calculi. If it seems advisable to give these drugs, they should be combined with sodium bicarbonate and given in small doses, such as 0.3 gm. (5 grains) two or three times a day. Large doses of atophan may cause gravel, and sometimes colic.

Normal urine should be acid, and to keep it alkaline for any length of time is generally bad treatment. Therefore alkalies should not be administered over an extended period of time as such procedure may result in cystitis, pyelitis or possibly the precipitation of phosphate calculi. Furthermore, any bacteria that may

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61. Haskins, H. D.: The Uric Acid Solvent Power of Urine After Administration of Piperazin, Lysidin, Lithium Carbonate and Other Alkalies, Arch. Int. Med., March, 1916, p. 405.

be lodged in the pelvis of the kidney or in the bladder, caused by the irritation of the previous calculus, will grow more rapidly in this alkaline medium.

If before or after the passage of calculus, or while a calculus is suspected or known to be located in the pelvis of the kidney, the urine is alkaline or ammoniacal without treatment, hexamethylenamin and dilute hydrochloric acid should be given as suggested above. If there is no marked bleeding from the kidneys or in the urine, the dose of hexamethylenamin should be 0.3 gm. (5 grains), dissolved in half a glass of water, four or five times a day, or 0.6 gm. (10 grains) in a glass of water, three times a day, for one or two weeks; the treatment should then be suspended, and perhaps later begun again. Very large doses of hexamethylenamin are not necessary. If the urine nevertheless remains alkaline, the hexamethylenamin is of no value, and one may conclude that the inflammation is serious, probably requiring surgical interference. With many bacteria in the urine the hexamethylenamin treatment is the correct one. Alkalies should not be given.

In some instances the pain is referred to the region of the gallbladder; in others, particularly in colic of the right side, to the appendix. Diagnosis in these cases is difficult, especially when not accompanied by hematuria.

Therefore, before operative interference is decided on for possible appendicitis or gallbladder trouble, roentgenograms should be made. It should be noted that not every calculus (even in the bladder) will cast a shadow; also that calcareous deposits in a tuberculous kidney may cast a shadow not unlike calculi. When there is doubt, and when the pain is a chronic, one-sided pain, particularly of the right side, cystoscopy and ureteral catheterization should be performed, and in some instances a dilating wax-tipped bougie may be passed into the pelvis of the suspected kidney. Pyelography may be utilized for diagnosis, as with a stone in the kidney it has been asserted that the pelvis is generally more or less enlarged. In acute cases without pus in the urine, and with a suspected appendix inflammation, a blood count showing a leukocytosis should indicate appendicitis. When gallbladder inflammation is suspected, there should be increased temperature, probably leukocytosis, staining of tissues with bile — which may also be found in the urine, and possibly distinct evidences of jaundice. In chronic cases in which there may be a stone in the kidney, and operative interference necessary, not only the foregoing examinations, but also functional tests of the kidneys, and blood tests for nitrogen retention should be made. If there has been a calculus in the kidney for some time, the function of that kidney is usually impaired, and there may be pus. If the stone is not passed within a month after the renal colic, radiograms of the kidney should be made. If no stone is thus located, it may be hoped it has dissolved or become disintegrated; if it is located, it should be surgically removed before the kidney is injured. The kind of operation to be performed on a kidney found to harbor a calculus depends on the condition of that kidney, and on the functional ability of the other kidney. This will be referred to later under another heading. Blood in the urine, more than a few corpuscles which may always be present, points distinctly to a pathologic condition in the urinary tract, and is rarely coincident with appendicitis or gallbladder trouble.

CALCULI IN CHILDREN

That urinary calculi occur in children is, of course, recognized; but a summary of the subject by Collins[62] shows that one investigator found "that 45 per cent. of 5,900 cases of calculus occurred in children, and that children under five years are especially prone to the condition." Collins finds that children resemble adults in that renal calculi occur in boys twice as frequently as in girls, the majority of cases being found in children under 2 years of age. Holt says that small renal calculi are very common in infants, and though these calculi may be voided without symptoms, other observers are of the opinion that renal colic in infants is not rare.

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[62] Collins, A. N.: Urinary Lithiasis in Infancy, Am. Jour. Dis. Child., October, 1913, p. 245.

The causes of these deposits in children are probably too much nitrogenous food, too much calcium in the food, perhaps too much phosphate in the food or medication, and often an insufficient amount of water. Perhaps frequently there are the added factors of bacterial infection and delayed passage of urine to the bladder. Gout in the parents can scarcely be called a predisposing cause. Bacterial infection, general and later focal, is doubtless a frequent cause. With bladder calculi, renal calculi may be presupposed. When blood or pus is found in the urine, a careful examination should be made.

Sudden pain, apparently abdominal (in a child too young to describe its symptoms), vomiting, and suppression of urine, or bloody urine, suggest a kidney stone; sudden high fever added to these symptoms indicates a kidney infection. A history of gravel, or of passage of urine showing a quantity of sediment, may aid in the diagnosis. Roentgenograms should be made before operative interference is undertaken.

It is very difficult to decide whether in young children morphin or codein would mask a necessary symptom in making a diagnosis — as in appendicitis or intestinal obstruction, for instance. At the same time, we are not justified in allowing a child to suffer agony when an adult would insist on the alleviation of his pain. Therefore when the renal colic has been diagnosed, the pain must be diminished. Hot baths may cause relaxation, with a dose of codein proportionate to the age of the child; a few whiffs of chloroform may also be needed. Long continued use of chloroform, however, is not justifiable.

A calculus which has not been passed should be sought by the Roentgen ray and surgically removed, as the lodging of such an obstruction in any part of the urinary tract is a greater menace to a child than to an adult.

The prognosis is unfavorable as many cases necessarily remain undiagnosed until necropsy. Surgical treatment of diagnosed cases, however, should give more satisfactory results.

The child who has passed a stone should be given the following prophylactic treatment: a properly balanced diet, regulation of the bowels, and plenty of water.

BILATERAL KIDNEY STONES

In children, as well as in adults, we frequently find that if stones are located in one kidney, the same condition will be discovered in the other. Speaking conservatively, this is true of 20 per cent. of the cases. This is necessarily true as any urinary or digestive disturbance has a tendency to affect both kidneys unless one kidney is made more susceptible by some localized predisposing cause. Hence, before operation both kidneys must be examined by the Roentgen ray. When stones are found in both kidneys, operation on either kidney is accompanied by greater hazard. Of course the more weakened either or both kidneys are from pus, hydronephrosis or nephritis, the graver is the prognosis. Whether or not the two kidneys in these bilateral cases should be operated on at once is a nice medical and surgical question; careful consultation, tests of function and of the blood for retention nitrogen must make the decision.

HEMATURIA

Hematuria, or blood in the urine in tangible amounts, should always be investigated, as it may be of serious import. Traces of blood, which are frequently found, may lead to the discovery of various disease conditions. Real hematuria may appear as clear blood or blood mixed with urine; or the urine may appear smoky, as occurs when the kidney substance is the source of bleeding, the blood being thoroughly mixed with urine, albumin and débris. Small amounts of blood may also be found in the urine in chronic nephritis of all types. Other conditions that cause or allow hemorrhage into the urinary canals are traumatism; calculi; cystic degeneration of the kidneys; carcinoma or tuberculosis of any part of the urinary tract; some blood diseases, especially hemophilia; acute infections of the malignant type; acute nephritis; septic infarcts of the kidney substance; and more rarely echinococcus infection. Blood may of course also appear in the urine owing to bleeding from the ureters, bladder, prostate or urethra. Rarely exposure to severe cold may cause blood to appear in the urine in certain individuals. Cardiac insufficiency, with passive congestion of the kidneys, congestion of one kidney due to displacement or to pressure on its blood vessel, a serious lordosis, or serious disturbances of the liver may cause kidney hemorrhage. Cases of hemorrhage have been reported in which there was localized dilatation of the blood vessels. This condition, which is rare, has been termed renal angioma or renal varix. Hematuria as well as hemoglobinuria may sometimes be caused by certain forms of pernicious malarial fever.

Elsner[63] found hemorrhage of the kidneys in 100 per cent. of acute tubal nephritis, in 100 per cent. of secondary congested kidney (passive hyperemia), in 33 per cent. of chronic tubal nephritis, and in 14 per cent. of chronic interstitial nephritis. These surprisingly high percentages give evidence of the frequency of hematuria, and should warn against operative interference except as a last resort. A floating kidney which is bleeding should be anchored, since a kidney so displaced as to cause interference with the outflow of blood from the renal vein must be passively congested.

It has been suggested that a kidney passively congested for some time becomes enlarged through dilatation of its blood vessels and is subjected to pressure from its capsule. Decapsulation would relieve this passive congestion and the resulting hemorrhage.

Blood issuing from the urethra will generally precede the passage of the urine or appear in the first jet. If it comes from the posterior urethra or from the prostate gland region, it is apt to follow urination. Blood coming from the bladder may be well mixed with urine, it may appear with strings of mucus, or it may follow urination. Blood from the ureter or from the pelvis of a kidney may follow urination, or the urine may be more or less mixed with it. As stated, blood thus occurring with nephritis generally causes a smoky urine, although clear blood may come from some lesion of the kidney substance.

Urethroscopic and cystoscopic examinations may disclose the origin of the blood. If its source is found to be in the kidneys, the passage of ureteral catheters will show the kidney from which it comes. Roentgenograms may reveal a calculus in the pelvis of a kidney or in the ureter as the cause of the hemorrhage.

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[63] Elsner: New York State Jour. Med., 1913, **13,** 144.

If the bleeding is due to a stone, absolute rest will usually cause a cessation. If it is due to tuberculosis — a common cause — or to some kidney growth, rest will probably not be efficacious; but, if the hemorrhage is localized in one kidney, surgical intervention may be required. Before resorting to this measure, however, one should take into consideration the health of the other kidney, the seriousness of the bleeding, and the gravity of the symptoms. If the bleeding is due to a displaced kidney, rest and abdominal support will usually remedy it. If it is due to circulatory insufficiency, cardiac tonics and rest will generally cure the condition, as whatever improves the system will of course diminish the hematuria.

Gallic acid has at times seemed to check renal hemorrhages due to disturbances of the blood or to a growth in the kidney. The use of gelatin and calcium chlorid has sometimes been beneficial, as has also been dry cupping over the offending kidney. Cotarnin hydrochlorid (stypticin) and cotarnin phthalate (styptol) have been used with apparent success in some cases of bleeding from the kidney substance. Injections of epinephrin solutions into the pelvis of the kidney through the ureteral catheter have suppressed bleeding for varying periods. Blood serum, the administration of suprarenal gland, or the administration of thyroid gland sometimes will prove effective when the hemorrhage is due to a hemophilic condition.

It should be emphasized that the most frequent causes of kidney hemorrhage are renal stone, tuberculosis and circulatory insufficiency. It is associated with acute nephritis. When accompanied by chills and fever, even though other evident sepsis is lacking, a septic infarct is probable. It should also be remembered that hexamethylenamin, which is now so frequently given, may cause hematuria. Occasionally blood vessel pressure from an abdominal tumor, or in pregnancy, may produce kidney hemorrhage.

ESSENTIAL HEMATURIA

Before applying this designation to a kidney hemorrhage, one must be certain that this particular condition may not come under some other heading. Kretschmer has said that essential hematuria is often a cloak for ignorance and the source of the bleeding should be carefully sought. Many unexplained hematurias are thus termed and Braasch[64] states that essential or unexplained hematuria is not a rarity. He found that 75 per cent. of seventy-seven cases were in males, the majority of whom were between 40 and 50 years of age. The duration of this intermittent hematuric condition varied, sometimes continuing for many years. Braasch found that high blood pressure was not a usual factor. These attacks, in some instances, were accompanied by pain. If the bleeding is preceded by a chill, if there is any pus or much albumin, a localized kidney inflammation is the probable cause of the hemorrhage. The presence of bacteria signifies perhaps a focal infection. Braasch found that in some cases injections of epinephrin solution, 1:2,000, into the pelvis of the kidney, while not curative, would stop the hematuria for a long time. He advises nephrectomy in persistently recurrent essential hematuria, when one kidney is healthy, having found nephrotomy of no value in this type of bleeding. It should be emphasized that before nephrectomy is employed in these cases, all less severe measures must be thoroughly tried. If bleeding then does not cease, the patient becoming exhausted and anemic, removal of the kidney is justifiable. Syphilis in some instances causes hematuria, and proper treatment of this disease may be the remedy required.

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[64] Braasch, W. F.: Clinical Observations on Essential Hematuria, The Journal A. M. A., Sept. 20, 1913, p. 936.

The pathology of these bleeding kidneys generally discloses the fact that the blood flows from the glomeruli, and that there is some kidney degeneration — a fibrosis not unlike that due to prolonged passive congestion or to heart insufficiency, as pointed out by Spitzer.[65]

It should be noted that, in many cases called "essential hematuria," plausible causes for the bleeding have later been discovered. It should also be noted that chronic nephritis may be present for a long time, causing hemorrhage even though the urine shows, on repeated examinations, no albumin and no casts. Such a condition may occur when there is arterial degeneration.

HEMOGLOBINURIA

In this condition the coloring matter of the blood appears in the urine without the corpuscles, the urine being dark and containing blood pigment and broken down red corpuscles. It may occur in diseases of the blood, such as pernicious anemia and purpura; in conditions causing the destruction of the red corpuscles, such as pernicious or malignant infections; in pernicious malaria, or in hemoglobinuric fever, and it may occur when nontested blood is transfused into an anemic patient or into a patient who has suffered hemorrhage. The latter incident rarely occurs at the present time, as it is now recognized that the donor's blood must be tested before transfusion can with safety be performed. Besides the causes just enumerated, a hemoglobinuria may be produced by poisons of the phenol type, such as coal tar products and potassium chlorate, and also by some snake poisons. Carbon monoxid (illuminating gas) poisoning is a frequent cause. Chloroform anesthesia may occasionally bring about this condition, as may also large doses of quinin. It is not necessary to discuss, in this connection, whether it is more hazardous to allow a pernicious malarial fever with kidney hemorrhage to run its course, or to decrease the fever by administering large doses of quinin, which will tend to increase the flow of blood.

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[65] Spitzer, W. M.: Continuous Painless Renal Hemorrhage and Its Treatment, The Journal A. M. A., Dec. 12, 1914, p. 2110.

Some time ago it was shown by Pearce, Austin and Eisenbrey[66] that there must be a concentration of hemoglobin in the blood of 0.06 gm. per kilogram of body weight before hemoglobin appears in the urine. If there are very large amounts of hemoglobin in the blood stream, bile pigments will also appear in the urine. A limited amount of the hemoglobin débris is cared for by the liver, but when this amount is exceeded the excess débris is secreted by the kidneys. Hemoglobinuria is frequently accompanied by hematogenous jaundice.

Paroxysmal hemoglobinuria is rare, but periodic attacks, with oxyhemoglobin and methhemoglobin in large amounts in the urine, may occur. Men seem to be more frequently afflicted with this form of hemoglobinuria than women. These attacks may be induced by cold, and in some cases by syphilis. They may be preceded by a chill and followed by a fever, with oft recurring myalgic pains. The urine will probably not continue hemoglobinuric longer than twenty-four hours. Malarial fever may be eliminated as producing such an attack; however, there usually is some hidden but definite cause.

PYURIA

Pus may appear in the urine in amounts varying from very small to large. A vaginal cause of pus in the urine in females can readily be excluded by catheterizing. In urethritis and cystitis the pelvis of the kidney is more frequently involved in the female than in the male, gonorrhea rarely ascending to the kidney in the male.[67]

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[66] Pearce, Austin and Eisenbrey: Jour. Exper. Med., 1912, **16**, 575.

The bacteria most frequently found in urine are the streptococci, staphylococci, gonococci, colon bacilli, pneumococci and tubercle bacilli.

C. Beeler and H. F. Helmholz[68] find, after examining 118 specimens of the catheterized urine of sixty-one female children, that colon bacilli are not normal inhabitants of the female urethra, but in extra-urinary infections occurring during the first two years of life the colon bacillus is frequently found in the female urethra; in girls over two years of age the urine is usually free from colon bacilli.

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[67] Sanders: New York Med. Jour., June 24, 1916, p. 1226.

[68] Beeler, Carol, and Helmholz, H. F.: The Bacteriology of the Urine in Health Children and Those Suffering from Extra-Urinary Infections, Am. Jour. Dis. Child. October, 1916, p. 345.

NEPHRITIS

CLASSIFICATION

Were it possible absolutely to accept Fischer's theory of nephritis, namely, that an overproduction of acid in the body or an accumulation of acid in the kidneys is the usual cause of this condition, many complex problems connected with it would become simple. If, as he believes, it were true that the edema is due to too much acid in the system, and that most of the uremic symptoms are due to cerebral edema, our understanding of the pathology and the treatment of these conditions would be clearer; but, unfortunately, the treatment which he sometimes recommends to overcome the supposed excess of acid may be hazardous. Water, as he states, is undoubtedly the best diuretic; but it cannot be accepted that no other substances cause diuresis. Carbohydrates in diabetics act as diuretics, while a number of drugs will cause diuresis in normal conditions of the kidney.

Perhaps the best classification of nephritis is that offered by Oertel:[69]

1. Simple nephritis.
2. Degenerative and exudative nephritis.
3. Degenerative and productive nephritis.
4. Productive nephritis.
    (*a*) Diffuse.
    (*b*) Focal and patchy.
5. Senile atrophy or nephritis due to arteriosclerosis of the kidney. Senile kidney.

1. The first class of nephritis is accompanied by all the classic symptoms of inflammation with exudate

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[69] Oertel, Horst: The Classification of Nephritis, Arch. Int. Med., June, 1913, p. 653.

and with desquamation and proliferation of parenchyma cells in some cases. Recovery may be complete.

2. The second class shows degeneration and necrosis exudate with hemorrhage, proliferation of parenchyma cells and formation of casts in abundance.

3. The third class shows not only degeneration and most of the signs and conditions of the second class, but also a production of fibrous tissue and shrinking of structure, with thickening of the renal arteries.

4. The fourth class is identified by the development of new kidney-secreting structure.

In the diffuse type of inflammation not only is new tissue formed, but also there is a growth of connective tissue and distortion of the tubules.

In the focal form, hyaline swelling and fatty degeneration occur in the intima of the small renal vessels, causing impaired function of the parts of the kidney fed by these blood vessels, or, in extreme cases, actual obliteration, marked by a granular appearance of the kidney.

5. The fifth class or arteriosclerotic kidney consists of a noninflammatory degeneration.

While the foregoing classification is based on pathologic findings and may be difficult to interpret clinically, it is scientific and recognizes the well known interlocking and overlapping cases of nephritis. An exact diagnosis of the pathology of a kidney cannot be made by the urinary or blood findings; we can determine the signs and symptoms and the amount of excretion and retention only at a particular date and hour. Frequently we cannot accurately prognose the length of life of the patient, even when he is moribund.

The old classification of parenchymatous and interstitial nephritis is not sufficient. Most parenchymatous nephritis would probably be more aptly termed diffuse nephritis. Probably no interstitial nephritis occurs without more or less parenchymatous degeneration, if not inflammation. Possibly the senile sclerotic kidney may be termed more or less purely interstitial. We may recognize the factors which tend to produce acute or chronic inflammation of the kidneys, and the factors which are most likely to cause a parenchymatous and diffuse or interstitial type.

ETIOLOGY

The facts that albumin and casts are frequently found in the urine in various ailments, and that in incipient and advanced nephritis acute disturbances of the kidneys frequently occur have not been better demonstrated than by the findings of Brown and Cummins[70] in the cases of 7,000 patients admitted to the San Francisco Hospital, the majority of whom were men between the ages of 20 and 60. They found albumin and casts in 594 patients, although only thirty-eight of these were admitted for nephritis. They found that venereal infections, the more serious general infections and alcohol played a prominent part in the previous history of chronic nephritis. They draw the logical conclusion that a kidney once injured is later more susceptible to irritations.

Localized nephritis is known to be caused by bacteria from some acute infection, pyemic in type, as with infarcts, or by a more chronic localization, as in tuberculosis. Localized or general nephritis may be caused by some hidden chronic infection, as mouth or nose infections; or a more or less acute nephritis may be the result of an acute general infection, such as scarlet fever, diphtheria, pneumonia, cerebrospinal fever or smallpox. All the above mentioned infections ordinarily are the sources of diffuse nephritis, perhaps primarily parenchymatous.

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[70] Brown, P. K., and Cummins, W. T.: A Study of the Etiology of Chronic Nephritis, The Journal A. M. A., March 11, 1916, p. 793.

Doubtless if the urine should be examined during the first stage of every acute infection, traces of albumin would frequently be found. The backache and high fever accompanying these infections is probably due to kidney congestion, caused by toxins of the bacteria, or by the disturbance of metabolism, especially protein. Probably, in the more simple general infections, the bacteria rarely reach or disturb the kidneys, but their toxins may affect these organs.

In lead poisoning, in syphilis, in prolonged cardiac insufficiency, in hypertension, in arteriosclerosis and in gout, although the nephritis may be more or less profuse, the interstitial form usually predominates. Prolonged intestinal disturbances, liver insufficiency, repeated strenuous physical labor, and polyglandular (ductless gland) disturbances are apt to cause chronic disturbance and chronic inflammation of the kidneys. Prolonged and repeated kidney congestion, due not only to cardiac insufficiency, but also to pressure from some abdominal cause, or to displacement of a kidney, tends to cause one or both kidneys to become infected with chronic inflammation. Probably abdominal pressure during pregnancy is a frequent cause of this type of nephritis.

The composition of the blood coming to the kidneys represents an important factor in the health of the kidney glands. The chief facts to be considered in this respect are: whether the blood contains the necessary nutriment; whether it carries elements causing dilatation or contraction of the kidney vessels; whether it carries an extra amount of waste products, drugs or foreign matter difficult of excretion or hard to metabolize or to change chemically into normal excretory products; whether the bulk of the blood coming to the kidneys is too great for the kidney structure to care for properly, or whether the amount of blood is insufficient to cause a proper kidney pressure both for nutrition and for excretion, making the kidney function normal. As stated, metabolic, organic or bacterial toxins may irritate or erode the kidney cells and sooner or later cause nephritis.

There doubtless is also a nervous control of the kidney circulation and of the kidney activity that at times plays an important part in an acute or chronic disturbance of the kidneys. Surgical conditions in the abdomen may reflexly interfere with the kidney function, making kidney tests inaccurate; this is especially true of ureteral, bladder and pelvic disturbances. Furthermore, disturbances of many glands, such as the liver, thyroid, suprarenals and pituitary, markedly change the work required of the kidneys.

As abnormal, repeated heart strain injures the heart and blood vessels, so repeated overmuscular effort, by increasing the exertion of the kidneys, may injure them.

Toxins or irritants absorbed from the intestine or from poisonous foods, cold storage foods, canned foods, or foods containing minute doses of boric acid or sodium borate may, by daily irritation, cause chronic nephritis. Chemical irritants such as turpentines, phenols and many aromatic oils or synthetic drugs, under certain conditions, may cause inflammation of the kidney. Too great an absorption of silver nitrate may cause acute nephritis, while prolonged use of mercury and arsenic may cause an acute or chronic form of this disease. Chloroform and ether may cause temporary kidney irritation.

The kidneys generally respond to an increased demand on them by a dilatation of their vessels with an increased general blood pressure. Following the ingestion of large amounts of protein or the circulation of irritants in the blood, an increased amount of blood flows through the kidneys in a given time and the excretion of urinary substances is increased; thus the blood is purified. The person who is repeatedly poisoned continues for months in apparent health, entirely unaware of the slowly developing chronic changes taking place in his kidneys. There comes, however, a day of reckoning; the kidneys, heart and blood vessels gradually work less harmoniously, and the kidney congestions result in chronic nephritis.

If the close relation of the kidneys to the skin, relative to excretions is considered, many unexplained albuminurias and attacks of nephritis will be understood. The excretion of solids by the skin is increased in this disease, although the total excretion is small.

While acute nephritis caused by cold alone is probably very rare, it may be the exciting cause of an already existing subacute or chronic disturbance. Chilling of the body at any time increases kidney activity, while warming of the body decreases it.

Any disease of the skin which prevents normal perspiration is detrimental to the kidneys, whether caused by burning, erysipelas, scarlet fever, dermatitis exfoliativa, or by any other acute or chronic general inflammation of the skin. The larger the area of skin affected, and the greater the inability to excrete water, the greater is the danger which threatens the kidneys. The reverse is also true: in chronic nephritis the immediate prognosis is more favorable when the skin is soft, moist and healthy, but is less favorable when the skin is dry and harsh.

Ether and chloroform anesthesia, especially the latter, which always causes kidney disturbance, by repeated use become a positive menace to the kidneys.

Diabetes may be a causative factor in nephritis, as frequently excessive amounts of nitrogenous food are consumed. Syphilis may also be an etiologic factor, either per se or through the arsenic or mercury treatment employed to cure it.

Chronic alcoholism is perhaps an indirect cause of nephritis since it disturbs the circulation, the liver and the digestion. Tobacco, by causing an abnormal blood pressure which finally injures the heart, becomes an indirect cause of nephritis.

The recuperative power of the kidneys and the harmless nature of occasional urinary symptoms of nephritis must, however, be recognized. Hence, extreme care should be used in diagnosing a case of nephritis. When a case is thus diagnosed it should be remembered that if one fourth of the kidney structure is healthy, this is sufficient to prevent serious symptoms. Furthermore, diseased parts may recover, or even if hopelessly degenerated, neighboring parts of the kidney may hypertrophy and carry on the extra work.

To sum up, heart and blood vessel insufficiency is a common forerunner of inflammation. This congestion may be the result of lordosis, or of a floating kidney. Prolonged exertion and hard work is a common cause of congestion and albuminuria, as is also profuse sweating, which increases the viscosity of the blood by dilating the kidney vessels.[71]

Prolonged low blood pressure and a reclining position associated with serious illnesses, such as typhoid fever and tuberculosis, are detrimental to the kidneys, resulting in extended passive congestion. Hence, the patient's position in bed should be changed frequently, just as soon as he is able to endure it.

Albuminuria, which frequently follows operations, may be due to the anesthetic, to an abnormal position long retained, such as that in pelvic operations, or to the two combined. Hence, anesthesia and abnormal positions on the operating table should be of as short duration as is compatible with efficiency and accuracy of technic.

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71. Sollmann: Am. Jour. Physiol., 1905, **13**, 24.

An important fact to bear in mind is that one attack of nephritis apparently breaks down the resistance of the kidneys, and that frequent attacks of albuminuria, which is generally caused by renal congestion, leaves them susceptible to real nephritis.

DISTURBED FUNCTION IN NEPHRITIS

Aschoff[72] states that the majority of primary contracted kidney cases develop as a result of vascular disturbance, "a sclerosis of the finest branches of the renal artery beginning relatively early in life and leading secondarily to the formation of atrophic foci in the parenchyma and to hyaline degeneration of the glomeruli, with a resultant focal inactivity and atrophy of the tubules belonging to them." The whole process is slow and passive. This slowly developing sclerosis of the fine arteries of the kidneys causing typical Bright's disease is not similar to the arteriosclerotic kidney of later life, when aortic disease precedes it, as in the latter case some parts of the kidneys may not be involved. It is self-evident that the more filter tissue incapacitated by arteriosclerosis the more efficient must be the circulation in the healthy parts of the filter. Hence the high blood pressure, cardiac hypertrophy and polyuria is nature's compensatory mechanism. Furthermore, the healthy glomeruli of the kidneys may hypertrophy so as to make the excretion from the kidneys sufficient without the assistance of the impaired sections.

In secondary contracted kidneys the previous history is that of an inflammation. In these cases the whole kidney filter is involved and the kidneys cannot react to any emergency demand on them; hence edema and uremia frequently occur. Usually a diminished amount of urine is passed, sometimes resulting in polyuria. In this condition the metabolism is the same as in primary contracted kidneys, namely, there is increased blood pressure, etc.

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[72] Aschoff, Ludwig: The Pathogenesis of the Contracted Kidney, Arch. Int. Med., December, 1913, p. 723.

Tests of function will show the ability of the recovered or still intact portions of the kidney to excrete, but do not show the excretory ability of the scar tissue. It has also been demonstrated by Baetjer[73] that in certain conditions of nephritis there may be not only a normal but also an increased eliminative power at some particular period. Hence one test, and even a repeated test with one substance, is not sufficient to demonstrate the health of the kidneys.

In the secondary contracted kidney the tubules connected with each diseased glomerulus gradually lose their power to function, to a greater extent than in primary contracted kidneys. The larger the number of glomeruli impaired or entirely incapacitated, the less the amount of urine excreted. The polyuria of hypertension and the consequent increased circulation in the unimpaired glomeruli should not be confounded with the polyuria caused by irritative toxins which injure the filter apparatus. Rapid impairment of normal function occurs in acute nephritis.

Oertel does not find that the amount of contraction of the kidneys bears any relation to the amount of cardiac hypertrophy.

Unless there is urea and nonprotein nitrogen retention in the blood, some other cause than kidney lesions is usually responsible for hypertension. Lead, once a common cause of contracted kidneys, is now of little importance ordinarily, as workers in lead exercise more care than formerly. Accidental chronic lead poisoning, however, from water pipes, hair tonics, etc., is still not of uncommon occurrence, although this cause of chronic nephritis, arteriosclerosis or other systemic disturbance is probably many times overlooked.

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[73] Baetjer, W. A.: Superpermeability in Nephritis, Arch. Int. Med., June, 1913, p. 593.

Ophüls[74] says that subacute and chronic glomerulonephritis, in the majority of cases, is due to chronic sepsis; hence the danger, long unrecognized, of chronic infection of the mouth, nose and adjacent sinuses. After the study of a thousand necropsies, Ophüls reaches the conclusion that the amyloid kidney, subacute and chronic (the so-called large white kidney) is also the usual result of chronic sepsis.

An interesting estimate of the general resistant power of the system in the nephritis of acute infections is suggested by Carles and Mauriac,[75] namely, that the more serious the infection, the more fragile the leukocytes; also that leukocytes break down more readily in nephritis than in health. This difference in their fragility in nephritis and in health, with the same technic, they find to be 50 per cent. Consequently, even a mild complication of nephritis in acute infection makes the prognosis grave.

Probably all poisons act primarily on the different parts of the convoluted tubules, the resulting variations in function being due to the parts of the kidneys most affected and to the effect on the blood pressure.

PRACTICAL INTERPRETATIONS OF EXPERIMENTAL NEPHRITIS

Uranium is perhaps the irritant most frequently used to produce experimental nephritis. Fitz[76] has shown that in acute uranium nephritis in rabbits the excretion of amylase is reduced and the nonprotein nitrogen of the blood is increased during the inflammation, but that the excretion returns to normal as the kidneys heal.

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[74] Ophüls, William: Subacute and Chronic Nephritis as Found in One Thousand Unselected Necropsies, Arch. Int. Med., February, 1912, p. 156.

[75] Carles and Mauriac: Jour. de méd. de Bordeaux, 1913, **43**, No. 18.

[76] Fitz, R.: The Relation Between Amylase Retention and Excretion and Nonprotein Retention in Experimental Uranium Nephritis, Arch. Int. Med., April, 1915, p. 524.

Mosenthal[77] has shown that all grades of nitrogen retention may be caused by varying the dose of uranium. With large doses the nitrogen excretion is diminished. With small doses the nitrogen not only may remain normal, but even may be increased. This he thinks may be due to the irritant causing a greater destruction of nitrogen in the body. He believes that a greater excretion of nitrogen by the intestine does not necessarily mean a vicarious elimination, representing, perhaps, only an increased nitrogen metabolism. The nitrogen output in the urine may be decreased even when the bulk of urine passed is sufficient. This seems to indicate that nitrogen is excreted by the convoluted tubules, but does not prove that the circulation of the glomeruli is not disturbed in uranium nephritis.

Underhill,[78] and later Underhill in collaboration with Wells and Goldschmidt,[79] showed that subcutaneous injections of sodium tartrate caused degeneration of the tubular epithelium. From later experiments he concludes that the convoluted tubules excrete urea, and that the glomeruli excrete chlorids and water.

Oertel,[80] in reviewing the subject of experimental nephritis, finds that when the sodium chlorid excretion is interfered with the urine concentration is low; when there is a diminished amount of urine excreted lactose excretion is slow, but sodium chlorid and potassium iodid excretion is normal, and in these instances the trouble is largely vascular; and when there is polyuria there is vascular hypersensitiveness.

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[77] Mosenthal, H. O.: Nitrogen Metabolism and the Significance of the Nonprotein Nitrogen of the Blood in Experimental Uranium Nephritis, Arch. Int. Med., December, 1914, p. 844.

[78] Underhill: Jour. Biol. Chem., **12,** 115.

[79] Underhill, Wells and Goldschmidt: Jour. Exper. Med., **18,** No. 4.

[80] Oertel, Horst: The Classification of Nephritis, Arch. Int. Med., June, 1913, p. 653.

More recently Myers, Fine and Lough[81] have studied the retention of creatinin, uric acid and urea in the blood in nephritis, and conclude that a uric acid retention without urea or creatinin retention shows early interstitial nephritis. Later in the disease urea is retained, and still later, creatinin. Kraus[82] had already shown that creatinin is more or less retained in acute uranium nephritis.

Frothingham, Fitz, Folin and Denis[83] found that in uranium experimental nephritis the excretion of phenolsulphonephthalein and the nonprotein and urea retention in the blood followed the course of the inflammation and returned to normal when the kidneys healed, and varied with the amount of kidney tissue involved. The recovery of the permeability of the kidneys to phenolsulphonephthalein may be rapid in this experimental nephritis, the test showing the condition of the kidneys at that particular period; but nonprotein nitrogen and urea may be much longer retained in the blood, and consequently the blood test for nonprotein nitrogen is a more positive indication of the condition of the kidneys.

Other poisons used in causing experimental nephritis or which accidentally cause acute irritant nephritis are chromium, mercuric chlorid, cantharides, croton oil, sodium arsenate and crotalus venom.

Without edema there is probably no retention of chlorids.

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[81] Myers, V. C.; Fine, M. S., and Lough, W. G.: Significance of Uric Acid Urea and Creatinin of Blood in Nephritis, Arch. Int. Med., April, 1916, p. 570.

[82] Kraus, W. M.: The Effect of Uranium Nephritis on the Excretion of Creatinin, Uric Acid and Chlorids, and the Effect of Creatinin Injections During Uranium Nephritis, Arch. Int. Med., June, 1913, p. 613.

[83] Frothingham, C., Jr.; Fitz, R.; Folin, Otto, and Denis, W.: The Relation Between Nonprotein Nitrogen Retention and Phenolsulphonephthalein Excretion in Experimental Uranium Nephritis, Arch. Int. Med., September, 1913, p. 245.

It seems to have been demonstrated[84] that, in experimental nephritis, diuretics, even the simplest, are irritant and harmful to the kidneys, and even if there is temporarily an increased output of urine and solids, there soon is a diminution of this output, and the lives of the animals are shortened. This coincides with the clinical findings that in most cases of nephritis, certainly in all cases of acute exacerbations in chronic nephritis and in acute nephritis, diuretics are harmful.

It seems to have been demonstrated that anything which increases the creatinin of the blood in nephritis, as muscular exertion or physical strenuosity, or infection or fever, adds to the danger from the kidney disease.

Brown[85] showed that hematin poisoning of the kidneys may occur during severe malarial intoxication, and that all grades of degeneration of the tubular epithelium may occur, with albumin, casts and blood in the urine.

Leopold and Bernhard[86] find that the nonprotein nitrogen of the blood, and the phenolsulphonephthalein test have the same values during health and in renal disease in children as in adults.

Zondek[87] believes, after carefully studying children with nephritis and with orthostatic albuminuria, that if the nephritis is of the vascular type and the glomeruli unable to excrete sodium chlorid and urea, the tubules assume this extra work. Hence, if the intake of salt is reduced, the tubules are better able to attend to excretion of nitrogenous substances; therefore, in glomerular inflammation, that is, vascular disturbances, it is not necessary to exclude nitrogen and salt from the diet at the same time. In other words, if proteins are temporarily excluded from the diet, a little salt may be allowed. Zondek found that by excluding salt the residual nitrogen of the blood was greatly reduced. These findings suggest variations in diet.

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[84] Christian, H. A., and O'Hare, J. P.: A Study of the Therapeutic Value of a Diuretic (Theobromin Sodium Salicylate or Diuretin) in Acute Experimental Nephritis, Arch. Int. Med., May, 1913, p. 517. O'Hare, J. P.: The Effect of Theobromin Sodium Salicylate in Acute Chromate Nephritis, ibid., June, 1915, p. 1053.

[85] Brown, W. H.: The Renal Complications of Hematin Intoxication and Their Relation to Malaria, Arch. Int. Med., September, 1913, p. 315.

[86] Leopold, J. S., and Bernhard, A.: Nonprotein Nitrogenous Constituents of Blood, and Phenolsulphonephthalein Test in Children, Am. Jour. Dis. Child., June, 1916, p. 432.

[87] Zondek: Ztschr. f. klin. Med., 1915, **72**.

O'Hare[88] has studied the excretion of sodium chlorid, nitrogen and water in nephritis. In normal individuals he found that on an increased sodium chlorid and nitrogen diet the urinary excretion became normal in forty-eight hours, but with diseased kidneys it may not return to normal in this period. He finds that the excretion of sodium chlorid generally is impaired in nephritis before there is much disturbance of water or nitrogen excretion. The excretion of nitrogen is greatly impaired only in severe cases. Excretion of sodium chlorid, water and nitrogen may be impaired in mild cases, even with normal phenolsulphonephthalein excretion and normal nonprotein nitrogen of the blood.

ANESTHESIA AND KIDNEY FUNCTION

It should be remembered that renal function as evidenced by one test of the phenolsulphonephthalein output is not sufficient, as this varies daily in a given patient. If the phenolsulphonephthalein was markedly retained, one test would be sufficient as indicative of the condition of the kidneys, as far as anesthesia was concerned. In advanced age the excretion of phenolsulphonephthalein is slower than normal, and this should be considered in operations on the aged.

Renal function as estimated by the phenolsulphonephthalein test has been found by Miller and Cabot[89] to be generally decreased in operations under ether, and is especially decreased after laparotomies. The decrease is best noted in the first twelve hours after the operation, and the more ether used and the longer the operation, the greater the decrease in the excretion of phenolsulphonephthalein, even to total inhibition in some instances. Shock (low blood pressure) caused a marked decrease. The lower the vitality of the patient, as from some serious condition like cancer, the more sluggish the phenolsulphonephthalein output. They found no definite relation of the decrease of phenolsulphonephthalein excretion to postoperative albuminuria.

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[88] O'Hare, J. P.: Study of Salt, Nitrogen and Water Excretion in Nephritis, Arch. Int. Med., June, 1916, p. 711.

[89] Miller, R. H., and Cabot, Hugh: The Effect of Anesthesia and Operation on the Kidney Function, as Shown by the Phenolsulphonephthalein Test, Arch. Int. Med., March, 1915, p. 369.

Slemons and Morriss[90] have shown that the rest nitrogen is increased in both maternal and fetal blood after chloroform anesthesia.

ACUTE CONGESTION OF THE KIDNEYS

Circulatory disturbances frequently occur in the kidneys. With every acute infection, with every high rise of temperature, with every extreme chilling of the body, and with every pathologic chill, the kidneys become more or less acutely congested. Chilling of the body will increase the amount of urine passed by raising the general arterial tension, and, if this amount is large, it will be of low specific gravity. Lowering of the general arterial tension, especially the surface tension, will diminish the amount of urine passed, and usually raises its specific gravity. During the high fever of all acute illnesses the amount of urine is generally diminished; it is usually concentrated and of high specific gravity, and urates and phosphates may be deposited on standing. It may contain albumin and hyaline casts. Lumbar backache is associated with the first stage of most acute infections, doubtless owing to kidney congestion. There are occasional cases of hematuria without special local pathologic condition.

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[90] Slemons and Morriss: Johns Hopkins Hosp. Bull., December, 1916, p. 343.

During the increased metabolism of high fever, the kidneys must do an additional amount of work to eliminate the increased waste products, as in a disease accompanied by high fever, much perspiration, or diarrhea, the amount of water excreted by the kidneys is diminished, thus requiring a greater effort in the excretion of salts. Therefore it is a physiologic necessity (unless some circulatory or stomach affection prevents) to give the patient a liberal amount of water during all feverish processes. If water cannot be retained by the stomach, it should be introduced into the colon.

If the skin is perspiring normally, the disturbance of the kidneys is less than when the secretion from the skin is abnormal; if the skin is dry or inflamed, as in scarlet fever, and is excreting little or no water, there is a strain on the kidneys. When the skin is affected by scarlet fever, erysipelas, large burns or other infections that interfere with its secretions, kidney irritation is a concomitant factor. When the skin is covered with perspiration, particularly if it is cold and clammy and if there is depressed circulation or shock, kidney excretion is generally diminished, and no treatment that does not improve the circulation and raise the blood pressure will be efficacious.

When the patient is suffering with edema or a more general dropsy, less water should be imbibed, and the treatment should be that of circulatory failure or nephritis, according to the cause of the condition. If an enormous amount of liquid has been lost from the body, whether by profuse watery diarrhea, persistent vomiting, diabetes mellitus, diabetes insipidus, or severe hemorrhage, water must be introduced into the tissues and circulation in the manner most effective for the preservation of life.

Ordinarily these acute congestions of the kidneys quickly disappear, and the kidneys again function in a normal manner. However, the cause of congestion may be greatly prolonged, as is the case in an eruptive fever with serious inflammation of the skin, such as scarlet fever. The irritation may then be prolonged until acute nephritis results, as the germ or the toxin of the germ of scarlet fever may be an added irritant to the weakened kidneys. In many acute general diseases, such as influenza and tonsillitis, the fever is short-lived and the acute congestion of the kidneys soon disappears. In more chronic infections, such as typhoid fever, the onset of the disease is often accompanied by a high fever; however, the temperature soon falls, and the slight kidney congestion apparently disappears, so that the only treatment necessary is to give the patient plenty of water.

Acute congestion of the kidneys may be caused by many drugs, such as the irritant diuretics, mercury, arsenic, potassium chlorate and antimony. Many coal-tar products of the phenol type, and synthetic drugs of the sulphonal type may cause irritation and even slight congestion of the kidneys. Acute nephritis may result from extreme intoxication produced by some of the more irritant poisons; kidney congestion may follow absorption of bacterial poisons from the intestine.

The symptoms of kidney congestion are backache and a frequent desire to urinate, while the small amount of urine passed is usually concentrated, with or without albumin, cylindroids, hyaline casts, and occasionally granular casts. Of course, many serious conditions of the kidneys do not cause backache, and furthermore, many lumbar pains do not have any relation to kidney disturbances; but it is believed that the lumbar myalgias associated with the beginning of acute infections, associated with signs of kidney disurbances, are due to kidney congestion.

CHRONIC (PASSIVE) CONGESTION OF THE KIDNEY

This disease is caused by interference with the return circulation in the renal veins; in other words, by any cardiac insufficiency which causes a damming back of the blood in the inferior vena cava, or by any pressure in the abdomen that causes interference with the circulation in the renal arteries or veins. This localized interference with the kidney circulation may be due to disturbances of the liver, ascites, tumors, pregnancy, lordosis or a loose kidney. Passive congestion of the kidneys may also be caused by lung conditions which hinder the return circulation through the inferior vena cava, such as pleurisy with effusion, emphysema, advanced tuberculosis, and tumors in the thorax. The urine in passive congestion is ordinarily concentrated, of a rather high specific gravity, tends to deposit urates on cooling, may contain albumin, and often shows blood corpuscles.

Chronic irritation of the kidneys often accompanies diseases of the liver, chronic diarrhea, or continued intestinal disturbance, such as chronic colitis. Chronic irritation long continued and chronic passive congestion will eventually cause kidney degeneration and chronic nephritis.

The only efficacious treatment for passive congestion of the kidneys is the removal of the cause. If this cannot be done, anything which will improve the circulation may, temporarily at least, improve the condition of the kidneys and increase the amount of normal urine.

ACUTE NEPHRITIS (ACUTE BRIGHT'S DISEASE)

Acute inflammation of the kidneys is usually caused by some acute infection, such as scarlet fever, diphtheria, measles, cerebrospinal meningitis, typhoid fever, smallpox, yellow fever, any septic infection, serious burns, erysipelas and acute general eczema, and by various poisons, especially the metallic poisons. Occasionally it begins as an inflammation without apparent previous cause, and in these cases it is supposedly due to cold; but cold alone does not ordinarily have such serious effect. The apparent onset may be sudden, edema, which generally develops in twenty-four hours, being the first symptom; or the inflammation may be preceded by a chill, followed by a slight fever. Nosebleed may occur, followed by hemorrhages into the retina, and if the amount of blood and albumin lost by the kidneys is great, anemia will rapidly follow. Almost similar symptoms indicate an acute exacerbation of chronic inflammation of the kidneys.

Inflammation of the kidneys is attended by the usual local symptoms accompanying any inflammation. There are an increased blood supply, swelling of the kidney, exudate into the tubules, and impaired kidney function; the urine diminishes and becomes concentrated, albumin, casts and blood appear, and frequently suppression is the eventual stage. Associated with these symptoms are headache, vomiting, and often diarrhea. There may be no fever, unless the acute nephritis is a complication of a condition that was already producing fever. There may be an increase or a decrease in the rate of the pulse; the former condition is the usual one. A certain amount of general edema occurs, its seriousness depending on the region of the exudate; edema of the lungs or edema of the brain may develop. The blood pressure generally rises, and with suppression of the urine, uremia with all its symptoms and dangers will probably result.

As stated above, when acute nephritis is a complication of some other infection or general condition, the symptoms will be modified by those of the causative disease; but when acute nephritis is the principal condition, if the patient is to recover, the kidneys begin to functionate, and a small amount of smoky, muddy urine, loaded with casts — hyaline, blood, epithelial and granular — is passed. Little by little the bulk of urine increases, the blood vanishes, the casts and albumin are diminished, edema disappears, and the patient slowly becomes convalescent. The excretion of chlorids, which have at first been almost absent from the urine, gradually increases as the edema lessens. The urine of acute nephritis is generally of a high specific gravity; however, the total amount may show a greatly diminished excretion of solids, while the blood retains nonprotein nitrogen.

If the nephritis is a complication of one of the exanthems, the skin may show the eruption of that particular disease; if the nephritis is not caused by an eruptive disease, it will probably be accompanied by urticaria, itching of the skin, and perhaps by a severe eczema. Forerunners of approaching uremia are eyeblurs, possibly retinal changes, severe headache, momentary losses of consciousness, muscle twitching and cramps, and eventually convulsions and coma.

When numerous cerebral symptoms announce the beginning of this disease the prognosis is very unfavorable and the patient may die within a few days; while in mild cases the inflammation may last several weeks. Whether the kidneys will again become entirely normal cannot be determined for several months, as small amounts of albumin and casts may persist in the urine for weeks and months, and yet the kidneys will finally return to a healthy condition.

The first intimation of an acute nephritis may be a general edema, with few other symptoms. Backache is not necessarily an accompanying symptom even in acute nephritis. This affection may also be superimposed on a chronic nephritis, due to some infection or to some carelessness in diet, or to exposure to cold.

TREATMENT OF ACUTE NEPHRITIS

The treatment of acute nephritis requires immediate bed rest in a warm, but properly ventilated, room.

During all acute diseases the urine should be frequently examined, and especially in diseases and conditions known frequently to result in nephritis, such as scarlet fever, erysipelas, serious burns, and poisoning, in order that the first indication of kidney inflammation may be noted. In these diseases all irritant foods should be prohibited, and all drugs which may cause irritation should be withheld, such as phenol compounds, synthetic drugs, irritant diuretics, aromatics, coffee, tea and alcohol. Observation of these cautions may prevent many kidney complications.

Cold applications should be used with great care; warm sponging is often as efficient as cold sponging in feverish processes. Whatever measures are taken in the treatment of the patient, chilling of the body should be avoided, although he should have fresh, cool air to breathe.

Baths should be taken in warm or hot water, as anything which promotes normal perspiration tends to relieve congestion of the kidneys; anything which tends to inhibit the secretions of the skin or to cause the skin to become cold and dry increases the congestion of the kidneys.

Meat proteins should be especially avoided as soon as traces of albumin are found in the urine, and the diet should become more and more rigid, according to the extent of the inflammation. The proper diet in acute nephritis is milk, or foods of the same character. If milk is not well tolerated, thin gruels, as oatmeal or malted milk, cereal milk, etc., may be substituted. Barley water is a good beverage and has some nutritive properties. Salt should be almost entirely removed from the diet. Nephritis patients usually suffer greatly from thirst. The amount of water advisable varies with the condition. Theoretically we should allow a considerable quantity, to wash out, if possible, the plugged-up tubules of the kidneys. Practically, however, if edema is extensive, much water should be avoided, although, if the circulation is efficient, pure water in a salt-free diet perhaps does not tend greatly to increase the edema. A refreshing drink may be prepared from a teaspoonful of cream of tartar in a pint of boiling water to which is added the juice of a small lemon, and a little sugar if desired. This beverage, when cold, is very agreeable.

If the patient's stomach is disturbed, a short starvation period is advisable. It is not expedient to continue this treatment long, however, as it tends to acidemia, which would add its quota to the toxins already more or less present. Liberal quantities of hot water may be given to relieve the vomiting, and if this is not sufficient, several 1 gm. doses of bismuth subcarbonate and sodium bicarbonate should be given every three hours until the nausea and vomiting cease.

The indications to meet are:

1. To promote elimination during the time the kidneys are insufficient.

2. To keep the waste metabolism and the metabolism of protein foods at the lowest limit possible.

3. To allay the inflammation, if possible, and to hasten the stage of resolution.

4. To prevent uremia if possible.

1. To meet the first indication the body should be kept very warm, and hot sponge baths should be given. Profuse sweatings are not indicated, except such as may be caused by hot drinks and warm applications to the region of the kidneys.

Unless contraindicated by some previous condition, an active cathartic should be given — a few grains of calomel, a saline purgative, or castor oil — to remove from the intestinal canal all substances which tend to the production of toxins which may be absorbed into the blood and increase the toxemia. If the patient is a child, rhubarb or cascara sagrada may be used. Daily movements of the bowels are necessary, but more than two or three loose movements a day are not indicated, and if the heart is weak, this amount of catharsis is often contraindicated. Enemas for purgation may be substituted for cathartics.

Diuretics should not be given, with the exception of pure water, or possibly a water containing some simple alkali, such as potassium or sodium citrate, or acid fruits which burn to alkali, such as oranges and lemons. The juice of these fruits is acceptable and beneficial to the patient.

2. To meet the second indication, the patient should be kept physically and mentally at rest. The more restless the patient, or the more muscle movements he makes, the more products of muscle metabolism reach the blood and must be excreted. Minimum quantities of all foods, particularly those containing protein, should be consumed, so that the kidneys may have the least possible amount of waste products to care for.

If the patient vomits, for a short time all foods should be withheld. The stomach may be washed out with hot water, or the patient may be given small doses of bismuth subcarbonate and sodium bicarbonate. However, starvation should not be too long continued, as it tends toward the production of acidosis and symptoms similar to uremia; therefore, bland, starchy foods should be given as soon as possible. If the stomach cannot retain them, alkalies, such as sodium bicarbonate or sodium citrate, in weak solutions, should be introduced into the colon.

If during the period of active nephritis the patient is restless and cannot sleep, it may be advisable to give a dose or two of chloral or of bromid, but synthetic drugs, or coal-tar products, or anything that forms hematoporphyrin during its elimination through the kidneys, such as sulphonal, trional, and drugs of that type, should be avoided.

3. The third indication is met by warm applications to the lumbar region, moist warmth in this region being of special benefit to acutely congested kidneys. Hot alcohol and water fomentations bound close to the back by a bandage around the abdomen, kept warm by a hot water bag, and changed as soon as cool, afford a simple method of applying such heat. The old-fashioned flaxseed poultice is also beneficial. Later the moist heat may be changed to the dry heat of an electric pad, which also brings about local hyperemia and in a reflex way gives relief to the kidneys. When the patient is kept warm, fomentations like those mentioned above cause perspiration, although not the profuse perspiration of the Turkish bath, caused by steaming, or by the introduction of dry hot air under the bedclothes. Such profuse sweatings are rarely necessary, and it is a question whether the good they have apparently accomplished was not due to relief of the kidney con-

gestion. Profuse sweating can do little more than eliminate from the blood large amounts of water, and the toxins retained in the blood in nephritis and in uremic conditions consequently become more concentrated. On the other hand, profuse perspiration sometimes causes the blood to absorb water from edematous tissues, which will counteract the concentration of the poison in the blood caused by the perspiration. If prostrating perspiration is caused by these applications, it is best to stop them and rely on the more modified heat of an electric pad.

This treatment, if successful, finally brings about an increase in the amount of urine passed, then the amount of blood in the urine is diminished, less albumin appears, although the urine may be loaded with casts, the edema and dropsy disappear, and the patient becomes convalescent, although edema may persist for weeks and albumin may not disappear from the urine for weeks or months. However, in kidneys that were normal before the attack of acute nephritis, the albumin and casts generally disappear in five or six weeks.

During this convalescent period the diet is gradually increased, although meat proteins and nuclein foods are still forbidden. In other words, if the edema has disappeared the patient is allowed bread, rice, more liberal amounts of cereals, and potatoes, but less milk. The amount of salt allowed him is gradually increased.

During this long period of abstinence from meat it is advisable to give small doses of iron, such as 5 drops of tincture of ferric chlorid, three times a day, in orangeade or lemonade; and after the albumin has disappeared from the urine, or after two months, although the albumin may not have disappeared, small amounts of meat once a day may be allowed, as a very long period of meatless diet is often not advisable in these cases. Eggs, simple vegetables and fruit should be added to the diet even before the albumin has disappeared; and when meat is allowed (meat includes fish, chicken and fowl) all beef extracts, beef broths and beef stock soups should be prohibited.

During this convalescent stage the patient may begin to sit up, walk around the room, and gradually about the house, but should not be exposed to cold, or allowed to attempt more than moderate muscular efforts. His bowels should move daily, and, if a drug is needed, aloin or cascara should be used. Children may be given milk of magnesia or some other simple laxative. It should be emphasized that a modification in diet is necessary according to the character of the digestion and the condition of the tongue. A liberal consumption of milk is often not well tolerated by adults, but a little careful investigation will determine the diet best adapted to the individual patient.

If the albumin and casts entirely disappear from the urine in six or eight weeks, the patient may be considered cured. However, severe exercise and chilling should be guarded against, lest the injured kidneys again be impaired.

If the albumin and casts continue, or if the twenty-four hour urine shows continued diminished excretion of salts or solids, although the blood may not show retained nonprotein nitrogen, the treatment of the case becomes that of a chronic nephritis.

Gentle massage may be employed as soon as the kidneys are functioning well, but it should not be resorted to if the secretion of the kidneys is insufficient. The amount of exercise the patient may take depends entirely on the efficiency of the kidneys in throwing off the waste material; if there is still danger of nitrogen retention, exercise should be limited. Warm baths and the milder hydrotherapeutic measures (no cold water and no chilling) are of advantage in keeping the skin in a healthy condition and in causing a good circulation of the blood on the surface of the body.

The prognosis of acute nephritis depends on its cause, but a case never should be considered hopeless. The patient may die, but with proper treatment he may recover, even from severe uremia, convulsions and coma. In acute nephritis there is no stage in which every effort possible should not be made to combat the disease and aid recovery.

A scarlatinal nephritis is more serious than that caused by many other conditions, and is apt to be insidious in development, occurring not only days, but weeks after the acute scarlatinal symptoms have subsided. Uremia may not develop even with temporary suppression of the urine; on the other hand, it may develop almost without premonitory symptoms, although ordinarily these symptoms appear hours or days before uremia occurs.

If the kidney secretion contains a great deal of blood, gelatin and lime water may be prescribed. Gallic acid at times apparently diminishes the loss of blood and albumin by the kidneys. If the blood pressure is low and the heart weak, congestion of the kidneys may be due to passive congestion, which may be alleviated by the administration of digitalis. Its action should be watched, as in impaired kidneys it may seriously overact, causing vomiting, headache, too high tension, and sometimes symptoms of uremia. On the other hand, it may be the drug required to relieve some of the congestion and to stop the bleeding. This type of acute nephritis, almost a passive congestion nephritis, not infrequently occurs in cardiac lesions, especially in valvular defects.

If the blood pressure is not very high, suprarenal gland extracts may be of value in stopping the loss of blood by the kidneys, especially in children. A crushed tablet, swallowed with water, every six hours for two or three days is a fair dose.

If the blood pressure is very high, nitroglycerin may be given in doses sufficient to reduce it somewhat; $\frac{1}{100}$ grain given once in three hours to an adult seldom fails to have this desired effect. If the hypertension in an adult is menacing, venesection may be employed; this may also be indicated if severe uremic symptoms, convulsions or coma are in evidence.

As several times stated, diuretics, as such, are generally contraindicated throughout the course of acute nephritis; even in the convalescent stage, if used at all, only the simplest diuretics should be administered with the greatest caution.

If the circulation is poor during convalescence, the amount of urine being insufficient, for this reason alone, and if digitalis is deemed inadvisable or inefficient, caffein in some form is the drug to be preferred, perhaps one of the best forms being theobromin and sodium salicylate, in doses of from 0.5 to 1 gm., in plenty of water. If the patient has reached the stage in which it is advisable to give caffein, there is no reason why a cup of coffee should not be given in the morning, or in the morning and at noon.

Throughout the course of acute nephritis, if the patient is comfortable he will probably sleep well. However, if he is nervous and irritable and cannot sleep, sleep must be induced in order that he may have the bodily and mental rest required. Synthetic hypnotics should rarely be administered, unless the dose is small, such as 0.2 gm. of veronal-sodium, or 0.05 gm. of luminal, since most synthetic drugs irritate the kidneys during excretion. If the patient is irritable, nervous and excited and the circulation is good, an adequate dose of chloral, 0.5 gm. perhaps, or 1 or 2 gm. of sodium bromid, may be used as a hypnotic, provided such strong drugs are deemed advisable.

Opium or any of its alkaloids are generally to be avoided in acute nephritis. However, if pain is acute it may be given, as acute pain can do more harm through circulatory shock, mental anguish and muscle restlessness than can possibly be done by a proper dose of morphin or codein.

If acute nephritis develops in the course of some severe infection, the treatment necessary to combat that infection should be given, modified in the manner suggested by the condition of the kidneys. All vaccine injections would probably be contraindicated.

As soon as the patient is convalescent, warm baths should be taken regularly, to keep the skin in perfect health and to relieve internal congestions. When convalescence is well established, if it is during the cold season of the year, the patient should, if possible, go to a warmer climate, and remain there until the kidneys have again resumed their normal activities.

4. The fourth indication, namely, the prevention of uremia, has frequently been met by proper management of the other indications. The treatment of this condition will be outlined later.

TREATMENT OF MERCURIC CHLORID POISONING

The first symptoms of mercuric chlorid poisoning are epigastric pain, nausea and vomiting, the vomitus sometimes containing more or less blood. The gastric symptoms may be almost entirely relieved by proper treatment, the success of which depends on the length of time between the ingestion of the poison and the treatment, and on whether the stomach was full or empty when the poison reached it. Diarrhea and signs of duodenal inflammation occur within a few hours, unless the poison was quickly vomited. If much mercury has been absorbed, stomatitis occurs on the second day.

If a tangible amount of mercury has been absorbed, there is soon a gradual diminution in the amount of urine passed, and after the first or second day there may be complete suppression. As noted in other acute metallic poisonings, total suppression of urine may not cause convulsions, as it ordinarily does in uremia, probably because some of the detoxicating functions of the kidneys may still be operative. After several days, delirium may occur, followed by coma, while the heart shows gradual failure.

If acute nephritis does not cause death, ulcerative colitis with hemorrhages from the bowels may prove fatal, even after a lapse of several weeks.

The following treatment of mercuric chlorid poisoning is founded on the therapeutic results and laboratory findings of Lambert and Patterson[91] and of Fantus.[92] It is best immediately to examine the first material expelled from the stomach either by vomiting or by lavage, and to examine the urine for mercury, as the patient may not have taken mercuric chlorid, as he imagines. The patient is first given the whites of several eggs; the stomach is then washed out. He is then given a pint of egg albumin water, and the stomach again washed out.

According to Fantus, a tablet composed of sodium phosphite, 0.36 gm., and sodium acetate, 0.24 gm., should be dissolved and administered as soon as possible. If this drug cannot be obtained, the following solution should be substituted: sodium hypophosphite, 1 gm.; water, 10 c.c.; hydrogen peroxid solution, 5 c.c. If the amount of poison swallowed is known, ten times that amount of hypophosphite should be given. As this dosage of hypophosphite might be large, it should immediately be followed by lavage with warm water and a greatly diluted solution of the antidote. This may be followed by a safe dose of antidote, which may be repeated every eight hours for several days. A glass of egg albumin water should also be imbibed every alternate hour until several doses have been taken. Since it has been shown that milk is worthless as an antidote, there seems to be no necessity for using milk, which curdles and causes acid. As a demulcent, starch water, slippery elm or flaxseed tea may be used. A gram of sodium acetate, dissolved in sweetened water, should be taken every three hours, for a day or two, being omitted at such times as it comes in conflict with the antidote.

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[91] Lambert, S. W., and Patterson, H. S.: Poisoning by Mercuric Chlorid and Its Treatment, Arch. Int. Med., November, 1915, p. 865.

[92] Fantus, Bernard: Jour. Lab. and Clin. Med., 1916, **1**, 879.

Lambert and Patterson's recommendation of rectal irrigation is important. They use the drop method of rectal irrigation, with a solution of potassium acetate, 4 gm. (a dram) to the pint. It might be well to substitute sodium acetate for the potassium acetate, as in all poisoning and in all serious conditions, sodium salts are safer than potassium salts, as this solution is more or less rapidly absorbed and generally causes diuresis. The colon should also be thoroughly irrigated twice daily to remove whatever mercury may have been there deposited or which may have reached the colon through the bowels. As there occurs a resecretion of mercury into the stomach, it should be washed out twice daily during the first few days unless the patient vomits repeatedly when given warm water. Lambert and Patterson affirm that it is advisable to continue the colonic drip enteroclysis day and night, at short intervals, even though it is intensely disagreeable to the patient, until two examinations of the urine on successive days have given a negative test for mercury. They also assert that if excessive doses of mercuric chlorid have been absorbed, or if the kidneys were previously diseased, the treatment should continue for a longer period, as long as three weeks, if necessary.

If the kidneys have become seriously involved and suppression is present when a patient first comes under observation, the prognosis, although very serious, is not hopeless. Lambert and Patterson advise a daily sweat in a hot pack. This treatment, and also frequently repeated stomach washings, depends on the condition of the circulation — if the heart is weak and the circulation poor, the hot pack may be inadvisable.

CHRONIC NEPHRITIS

Some of the causes of this disease are:

Unresolved acute nephritis.

Repeated attacks of acute nephritis.

Repeated irritations of the kidneys, such as result from repeated anesthesias, athletic strain, and frequent severe chilling of the body.

Overindulgence in meat and canned foods, repeated ptomain poisoning, and prolonged use of foods preserved with sodium benzoate or boric acid.

Intestinal indigestion.

Overindulgence in alcohol, especially beer. (Although alcohol has not been proved to be a direct cause of nephritis, it acts as a predisposing cause by disturbing the gastro-intestinal digestion and the liver.)

Prolonged hypertension.

Cardiac insufficiency.

Pregnancy.

Abdominal tumors.

Loose kidney.

Syphilis.

Kidney infection, especially of the pelvis of the kidney.

Gout.

Arteriosclerosis.

Chronic lead poisoning.

Long continued use of mercury, arsenic, some synthetic drugs, and some coal-tar drugs.

Repeated exposure to severe cold.

SIGNS OF INCIPIENT CHRONIC NEPHRITIS

Besides the more scientific tests of kidney function, many clinical signs and symptoms indicate the beginning of or the later stages of chronic nephritis. Acute nephritis invariably, and chronic diffuse nephritis generally, present so many urinary evidences that the diagnosis is unmistakable. The so-called chronic interstitial and arteriosclerotic nephritis begins and develops so insidiously, at times without urinary signs, that associated symptoms and conditions are of importance in making a diagnosis. Predisposing causes are: chronic congestion of the kidneys due to insufficiency of the circulation or to some abdominal cause; continued intestinal indigestions, as evidenced by indican in the urine; hypertension with all of its associated symptoms, as attacks of polyuria, arising at night once or more to urinate, cardiac hypertrophy, attacks of palpitation, slight dyspneic attacks, momentary dizziness, and eye disturbances; gouty symptoms; a history of overfeeding, especially of proteins; an alcoholic history; excessive smoking; severe athletic work or other physical tension in earlier life; previous attacks of nephritis; repeated pregnancies, especially if kidney congestion has occurred during this condition; and history of serious infection or disturbance of the urinary tract. A previous history of any of these conditions demands that the urine be examined at regular intervals for albumin and casts, and that the twenty-four hour urine excretion be determined. As chronic nephritis often develops slowly, proper advice and care will, in many instances, long postpone insufficiency of the kidneys.

The importance of the diminution of the chlorid excretion is understood, as is also the importance of preventing intestinal indigestion and of prescribing food which does not cause indican or an increase of sulphate to appear in the urine. If there is an increase of ammonia in the urine it may indicate liver disturbance.

The diet being arranged for the individual, repeated chilling of the body prevented, severe exercise proscribed, alcohol prohibited, tobacco prohibited or allowed in moderate quantities, the skin kept moist and healthy and able to function normally, and constipation prevented, incipient nephritis may for a long time remain in this stage.

On the other hand, as repeatedly stated, traces of albumin and hyalin and granular casts do not necessarily denote that chronic nephritis is developing. They denote some congestion of the kidneys, however, the cause of which should be sought, as these repeated congestions or irritations lead to future chronic inflammation. Even when an albuminuria, with or without casts, persists for some time in a condition not severe enough to be termed a nephritis, the congestion may entirely disappear, the injured epithelium entirely recover and the kidneys become normal, remaining so perhaps for years or for the rest of the patient's life. It seems probable that slight congestion of the glomerular epithelium may permit an albumin leak, while considerable degeneration of the epithelium of the tubes may occur without accompanying albuminuria.

Some portion of the kidney may be the source of considerable albumin and numerous casts and yet may entirely recover without interfering with the total kidney excretion or without showing symptoms of a real nephritis. The extent of the infection and the probable prognosis can be ascertained by tests of function, several twenty-four hour urine examinations, the condition of the circulation, the blood pressure and other associated symptoms, especially head symptoms, and also by an examination of the blood for nonprotein nitrogen retention.

PROGNOSIS IN CHRONIC NEPHRITIS

The height of the blood pressure is not a definite guide as to the severity of nephritis, but is an important indication, as a low blood pressure is rarely found when the kidneys are insufficient; though it may occur when the kidney inflammation is secondary to cardiac insufficiency. Although the contrary has been repeatedly asserted, cases have occurred in which the systolic blood pressure has been over 200 mm. and there has been no kidney insufficiency nor has one developed in months, or perhaps years. Overstimulation of the suprarenals by some hormone or by some disturbed excretion from the kidneys in nephritis may cause high blood pressure. Hypertension may be due to a certain extent to retained water and solid constituents in the blood in nephritis, this overdistention causing increased vascular pressure and perhaps compensatory hypertrophy of the heart. These retained substances in the blood may also irritate the walls of the blood vessels to contraction.

Although repeated examinations of the urine will almost invariably show, sooner or later, albumin or casts, or both, uremia may occur when they are entirely absent. Generally, however, the greater the number of casts and the greater the amount of albumin, the more severe the chronic nephritis. Large amounts of albumin may occur in orthostatic or lordotic albuminuria without a real nephritis.

A more positive indication of the condition of the kidneys than the amount of albumin and casts is the amount of nonprotein nitrogen in the blood. One hundred c.c. of blood should normally not contain much more than 30 mg. of nonprotein nitrogen. In uremia there is always retained nonprotein nitrogen, and in sudden coma in patients not previously observed when the urine apparently is negative, or when there is some suppression, examination of the blood for retained nitrogen will determine whether nephritis is a cause, or an associated cause, of the coma. However, the type of nephritis cannot be diagnosed by the amount of retained nonprotein nitrogen.

As elsewhere stated, the phenolsulphonephthalein output—50 per cent. of which should be normally excreted in two hours — may be normally excreted even in the presence of a moderately advanced nephritis. On the other hand, if the phenolsulphonephthalein is very slowly excreted, the kidneys are markedly impaired, either temporarily or permanently. In congestion caused by circulatory failure this impaired function may gradually be restored as the circulation improves. A diminished sodium chlorid excretion can be ascertained by testing, but may be inferred to accompany edema. If the kidneys are seriously impaired, nitrogen retention, as shown by the examination of the twenty-four hour urine on a known diet, or by the urea feeding test, is of serious import, although the type of nephritis cannot be determined by this test.

Evidences of kidney insufficiency other than the signs described are mental irritability, headaches, dizziness, periods of depression, increased muscle tension, an unexplainable fatigue, and weariness without much exertion, momentary deafness or blindness, or more serious affections of the eyes, and momentary attacks of aphasia. The mental powers may be seriously impaired, the patient finding it difficult to concentrate; memory and judgment may be impaired. These symptoms are characteristic of general arterial or cardiovascular-renal disease. More serious mental depression, approaching melancholia, may signify chronic nephritis.

Acidosis apparently is a frequent cause of dyspnea in nephritis; that it is the only cause is probably not true. Peabody,[93] who studied the relation of acidosis to dyspnea in kidney disease, concludes that a mild acidosis in chronic nephritis is compensated by the increased urinary output of acid. In uremia, acidosis is present, and in terminal uremia it occurs in a marked degree. He did not find that acidosis bore any relation to the nonprotein nitrogen retention or to the phenolsulphonephthalein output. Occasionally acidosis may be present without dyspnea.

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[93] Peabody, F. W.: Studies on Acidosis and Dyspnea in Renal and Cardiac Disease, Arch. Int. Med., August, 1914, p. 236.

Exophthalmos may occur in chronic nephritis, owing perhaps to some toxin acting on the sympathetic nervous system.

Chronic nephritis may be so insidious that the patient may apparently be well for years during the slow development of the disease, until retinitis, cardiac failure, apoplexy or uremia suddenly supervenes. Others, with or without albumin in the urine, may succumb to a sudden fatal attack of angina pectoris. Some patients suffering with chronic nephritis may develop the following symptoms: anemia, loss of weight (unless there is marked edema), low blood pressure, lack of strength, followed by severe headaches, resulting in death from acute uremia; others may have practically no head symptoms, and succumb to dropsy and heart failure.

SYMPTOMS IN CHRONIC NEPHRITIS

An acute diffuse nephritis, in which the parenchyma of the kidneys is most seriously affected, produces somewhat different symptoms than a chronic diffuse nephritis in which the interstitial tissue is most involved. As previously stated, it is better to consider the pathology of chronic nephritis than to consider parenchymatous and interstitial nephritis as two different types of kidney disease, as neither type occurs alone.

The general symptoms of chronic nephritis may be thus tabulated:

1. Changes in the urine as to frequency, color, specific gravity and abnormal content.

2. Headache, generally occipital, but often frontal, general or hemicranial.
3. Indigestion, with perhaps nausea and vomiting.
4. Diarrhea attacks preceded and followed by constipation.
5. Mental apathy or great irritability.
6. Insomnia, in a later stage of the disease followed by somnolence.
7. Dyspnea.
8. Edema of the face, hands or feet (not always present).
9. Intermittent eye and ear disturbances.
10. Enlargement of the heart (hypertrophy, dilatation).
11. High blood pressure.
12. Neuralgias.
13. Anemia.
14. Retinal change.
15. Dropsy or marked localized exudate (many patients show no edema or dropsy).
16. Terminal inflammations (pericarditis, etc.).
17. Uremia (convulsions and coma).

The amount of urine passed varies with the different forms of nephritis; in interstitial nephritis, or so-called contracted kidney, with high blood pressure, typical Bright's disease or cardiovascular-renal disease, the amount of urine passed in twenty-four hours may be large, especially the amount passed during the night. A marked diminution in the amount of urine passed, especially if the specific gravity is low and the excretion of solids and toxins is not sufficient, causes headache, restlessness, insomnia, indigestions, myalgias, muscle twitching and, if the excretion is sufficiently diminished, symptoms of uremia. Suppression of urine, however, may last several days without uremia. On the other hand, uremia may occur in chronic contracted kidneys even when a large amount of low specific gravity urine is passed. In the early stages of chronic diffuse nephritis the urine may be very scanty.

It may be highly colored and of high specific gravity, loaded with albumin and casts, and frequently contains traces or even tangible amounts of blood. In chronic diffuse (parenchymatous) nephritis, all varieties of casts may be found—hyaline, epithelial, granular, fatty and waxy.

Every type of headache, a feeling of fulness in the head, dizziness, ringing of the ears, irritability, restlessness, insomnia, loss of memory, stupidity or actual somnolence may occur in different stages and in the different types and conditions of chronic nephritis. Head symptoms may be the first indications of this disease, especially of the interstitial variety. The patient may awaken every morning with a headache or headaches may recur as acute attacks, accompanied by vomiting, a diminished amount of urine, and generally an increased blood pressure. Cerebral apoplexy may occur with any type of chronic nephritis, but it is most frequent in the interstitial variety.

Dyspnea is a very frequent symptom in chronic nephritis. It may be due to disturbance of the cerebral circulation, slight acidosis, hypertension or cardiac disability.

Gastric indigestion is usually a symptom of chronic nephritis. This condition is accompanied by anorexia with a special distaste for food in the morning. The patient complains of a bad taste in the mouth, especially noticeable in the morning, and of a bad tasting fluid which collects in the mouth during the night. The tongue is more or less coated, and often typically coated in irregular patches with small regions from which the coating is absent. This condition of the tongue is typical, though not pathognomonic, of advanced chronic nephritis. There is more or less distress after eating, with flatulence, pyrosis, nausea or vomiting, according to the ability of the kidneys to excrete and to the sufficiency of the circulation. Insufficient circulation with damming back of blood in the portal system, combined with liver congestion and impaired kidney function, causes gastro-intestinal indigestion which is hard to combat. Frequently vomiting resulting from this condition is one of Nature's methods of relieving the system of toxins, the kidneys being insufficient.

Irregular attacks of diarrhea, not excused by mistakes in diet or changes of temperature, often occur in chronic nephritis. These attacks usually occur when the amount of urine is scanty or the excretion of solids is diminished, and is another of Nature's methods of eliminating a certain amount of toxic products. This relief of vicarious excretion, however, is only a momentary help to the system. On the other hand, constipation is a menace in chronic nephritis, as congestion of the portal circulation, and consequently of the liver and stomach, may have serious results. However, while constipation should be relieved, profuse purging, except occasionally, is not advisable, particularly if the heart is weak.

Edema is usually first noted in the face, in the form of puffing under the eyes in the morning, which disappears after the patient has been up for a while; sometimes one or both hands are swollen in the morning, and there is perhaps some swelling of the hands or feet in the late afternoon and evening. Backache, perhaps due to edema of the tissues in the lumbar region, may be evident in the morning. In the later stages of the disease edema may be present in any part of the body, located either symmetrically or asymmetrically. It may persist all day, general anasarca may develop, and there may be an exudate into various cavities of the body. On the other hand, when the interstitial type of nephritis is predominant and the heart is sufficient, the disease may progress to uremia and death without evident edema.

The eyes may show numerous symptoms and signs in nephritis. There may be eye-strain headaches, momentary eye blurring, or things may "turn black before the eyes," as the patient describes the condition, such blurring lasting but a moment. There may be neuroretinitis or hemorrhages into the retina, that is, typical nephritic retinitis; and there may even be fatty degeneration of the retina. Albuminuric retinitis, which simply means retinitis associated with albuminuria, is a serious symptom and foreshadows an unfavorable development of the case.

The patient frequently experiences ringing in the ears or partial loss of hearing, probably most frequently due to arterial degenerations or to high blood pressure, and less frequently to central brain disturbances or to some nerve lesion in the internal ear.

If chronic nephritis develops rapidly, the heart may not be much hypertrophied, and cardiac weakness may be evidenced from the beginning. If chronic nephritis develops slowly — unless the heart is degenerated or has been impaired by some lesion — the left ventricle, especially, becomes greatly enlarged in its effort to overcome the increased resistance in the arterial system and the increased blood pressure. This is Nature's method of compensation to compel enough urine to pass through the diseased kidneys to effect sufficient elimination. It can readily be seen that the more slowly chronic nephritis develops, the more circulatory compensation will develop. In cardiac disease, especially in valvular lesions, with long continued sluggish circulation in the veins, particularly of the abdomen, congestion of the kidneys and a slowly developing diffuse nephritis occur. We then have a chronic inflammation of the kidneys superimposed on the heart lesion. This type of kidney disturbance will often greatly improve, even when apparently serious, with treatment aimed to improve the circulation.

A frequent type of chronic nephritis, with the interstitial type predominating, is that which is now recognized as cardiovascular-renal disease, or true Bright's disease. This slowly developing insidious nephritis is preceded by a long period of hypertension and cardiac hypertrophy, and is soon associated with more or less chronic myocarditis. The time to impede the progress of this disease is during the stage of previous hypertension. The manner of its final termination — whether by apoplexy, angina pectoris, cardiac dropsy, or uremia -- depends on which of the three organs affected is most diseased. The danger to the heart and to the cerebral arteries through high blood pressure is well understood, and the necessity of generally lowering hypertension, whether or not the kidneys are much involved, is now generally recognized. A systolic pressure of over 150 mm. at any age should cause the patient to be carefully examined to determine whether or not an attempt to reduce this pressure would be wise. A systolic pressure of over 200 mm. has long been supposed to indicate kidney disease. This is not always true, but it is a pretty safe working rule to suspect the kidneys when the pressure is at such a height., The most significant indication of kidney insufficiency is a diastolic pressure of 120 mm., and if that pressure is over 110 mm. there must be serious doubt as to the condition of the kidneys. Any diastolic pressure of 110 or over should be reduced, if possible. If the diastolic pressure is below 100 mm., even if the systolic is 160, there may be no need of attempting to lower the pressure, but a systolic pressure above 170 requires such an attempt to be made. If a previous high pressure becomes suddenly diminished without treatment, myocardial weakness has developed and more or less dilatation will soon be in evidence. If treatment causes too great a drop in blood pressure, especially in the systolic pressure, the treatment is too strenuous, and the patient feels weak and incompetent.

A frequent condition in chronic nephritis is pain in one or more nerves, and an actual neuritis may be experienced. These pains probably are frequently caused by an imperfect elimination of irritants or toxins from the blood, and some particular nerve or nerves subjected to injury, muscle strain or occupation tire is the first nerve or set of nerves to be affected. Before any local treatment is of much avail, the kidneys and blood must be in the best possible condition. If the patient is uremic, neuralgic pains may be so intense as to require hypodermic injections of morphin.

There probably will not be much pain in the region of the kidneys in chronic nephritis, although there may be a backache resulting from some edema in the tissues of the back, which will be especially noticeable on arising in the morning. This kind of backache usually disappears soon after the patient arises. A pain in the kidney itself is probably due to a surgical condition, such as a calculus, tumor, or some distention of the pelvis of the kidney.

The stage in chronic nephritis at which anemia will occur depends on the amount of albumin lost, the oozing of blood from the kidney, and the disarrangement of digestion and assimilation. Besides the direct loss of nutriment from the blood and the direct loss of blood corpuscles, the toxins circulating in the blood probably interfere with the nutrition of the bone-marrow, and the red blood corpuscles are probably not produced in normal numbers, or at the normal rate. In the treatment of chronic Bright's disease, thoughtlessness is frequently displayed in not protecting the blood from deterioration. To relieve the irritation of metabolic substances in the kidneys, and other signs of toxemia, too much purging is resorted to, and a rigid diet (withdrawal of all meats) too long continued. If it is advisable not to allow a patient meat, iron must be given, and he should receive enough vegetable proteins to keep up his normal metabolism; otherwise he will become anemic from the treatment alone.

Terminal inflammations are liable to be associated with uremic conditions. By a terminal inflammation is meant some associated condition which would probably not cause death under ordinary conditions, but which, when associated with chronic nephritis, is dire and fatal. A simple infection like tonsillitis, grip, a slight infection of the bladder, slight prostatic disturbance, and acute infections, such as pneumonia, may rapidly increase the seriousness of chronic nephritis, and the patient may die of uremia or of a combination of the two conditions. Edema of the lungs in these associated conditions is of frequent occurrence. Pleural exudate, pericardial exudate or ascites may rapidly develop with the fever process. The amount of urine excreted may rapidly diminish and uremia develop, whereas if the acute condition had not occurred, the chronic nephritis may have continued slowly advancing for years.

Besides the cerebral symptoms already mentioned, there is occasionally great mental depression verging on melancholia, and frequently excessive irritability and a discouraged and pessimistic frame of mind. There may be loss of mental fixation, poor memory, slight momentary attacks of aphasia, lack of good judgment, and a tendency toward repetition. These patients are often easily fatigued by either physical or mental activity. These symptoms usually occur with the arteriosclerotic or senile kidney.

The relation of acidosis to dyspnea has received considerable study. Some investigators believe it is a prominent cause of dyspnea. Acidosis, however, is not the only cause of dyspnea in cardiorenal disease; nor is this disease necessarily accompanied by dyspnea. Furthermore, the amount of acidosis does not seem to be related to the nonprotein nitrogen retention.

AMYLOID KIDNEY

The amyloid kidney was described as a separate entity by Rokitansky seventy-five years ago. It is now considered an amyloid degeneration occurring in chronic nephritis, particularly when the nephritis results from some chronic infection. It is found in the nephritis of chronic suppuration, and it may occur in tuberculosis of the lungs, bones and intestine, in syphilis, in malignant growths, and in some blood diseases, such as leukemia. The urine may remain normal in quantity or may be diminished. It is usually pale, of a specific gravity from 1.010 to 1.020, with large quantities of albumin, and the typical waxy looking hyaline casts.

ARTERIOSCLEROTIC KIDNEYS

In this form of nephritis the fine renal vessels show the characteristic changes of sclerosis. The kidney withers[94] as contrasted with granular kidney, the latter being caused by a slow irritative process, though both kinds of kidney trouble show focal distribution. In granular (contracted) kidney there is localized arteriosclerosis entirely separate from any general arteriosclerosis, while in senile (arteriosclerotic) kidney the disease of the kidney vessels is a part of the general arteriosclerosis. Senile kidney, though insufficient for added work, physical or dietary, and often insufficient in general infections, in ether anesthesia, and in other temporary conditions, may be entirely sufficient for a long time when the patient leads a calm, uneventful life.

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[94] Allbutt, C.: Brit. Med. Jour., April 15, 1911, p. 853.

It must be understood that the kidneys grow old with the rest of the body; hence a senile contracted kidney is not pathologic, unless it occurs at too early an age. This type of kidney is accompanied by slight albuminuria, casts, and often by a diminution in the amount of urine.

Numerous varieties of acute, subacute and chronic parenchymatous and diffuse nephritis may develop in senile kidneys, as they are very susceptible to infection. Elderly men whose kidneys may be only slightly impaired but in whom an enlarged prostate may cause damming back of the urine may suddenly develop uremia. Unless seriously contraindicated, such an enlarged prostate should be operated on, and life may thus be prolonged, even though the kidneys are arteriosclerotic.

EDEMA

In kidney disease the water and the solid constituents of the blood are more or less increased. The blood vessels being thus overfilled, the irritants in the blood stimulating the arterioles to contraction, and the heart becoming hypertrophied, the blood pressure rises. Eventually the increased permeability of the capillary walls causes the water and salts to be exuded into the surrounding tissues. The tissues most affected by such exudates are those in which the external or surrounding pressure is the least, that is, those which are most dilatable, and those situated where the circulation is the least forceful owing to the distance from the heart, or where the return circulation is sluggish. It has recently been shown by Underhill and others[95] that in chronic nephritis the blood vessel walls may not readily pass nutriment to the tissues or absorb waste products and water from the tissues.

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[95] Bogert, L. J.; Underhill, F. P., and Mendel, L. B.: Studies of the Permeability of Cellular Membranes, I, The Regulation of the Blood Volume After Injections of Saline Solutions, Am. Jour. Physiol., 1916, **41**, 189; II, The Action of Saline-Colloidal Solutions upon the Regulation of Blood Volume, ibid., p. 219; III, The Influence of Alkaline-Saline Solutions upon Regulation of Blood Volume, ibid., p. 229.

It should be recognized that in circulatory disturbances of the kidney vessels, either central or local, edema of the kidney substance may occur, and that after death the kidneys may be heavy with this infiltration.

Fischer's acid theory of edema has not been generally accepted; consequently, the forced feeding of salt and alkali is not indicated. A large amount of sodium bicarbonate may produce edema by causing chlorid and water retention, especially when as much as 20 gm. or more a day of sodium carbonate are given. This fact has been noted when large amounts of alkali have been employed in diabetes.[96]

Pearce[97] showed that in uranium experimental nephritis the administration of much water caused edema, and that arsenic caused edema in chromate nephritis. He concluded that three factors operated in producing edema, namely, injured kidneys, vascular insufficiency, and too much water in the blood. Hess[98] reports, from a study in the medical clinic of Vienna, that injury to the blood vessels is an important factor in dropsy.

As far back as 1902, Hatcher and Sollmann[99] found that chlorids were greatly diminished or absent in the urine in infectious diseases, and of course were entirely absent in salt starvation. The disappearance of the chlorids from the urine does not cause any great change in the other urinary constituents. They believe retention of chlorids in infection shows that the body requires more sodium chlorid than it is receiving from the diet; hence, as the patient should receive about 15 gm. a day, they suggest the addition of salt in a nearly salt-free diet, such as a milk diet.

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[96] Levison, L. A.: The Occurrence of Edema from Large Doses of Sodium Bicarbonate, The Journal A. M. A., Jan. 23, 1915, p. 326.

[97] Pearce, R. M.: The Production of Edema, Arch. Int. Med., June, 1909, p. 422.

[98] Hess: Ztschr. f. klin. Med., 1915, **82**.

154. Sollmann: Ibid., pp. 155-174.

[99] Hatcher and Sollmann: Am. Jour. Physiol., Dec. 1, 1902, pp. 139-

An insufficient salt diet such as is so frequently employed during fever, causes a resorption of chlorids from the tissues into the blood. As Sollmann and others have shown that sodium sulphate injected intravenously causes a disappearance of the chlorids from the urine, cathartic salts, especially sulphates, apparently should not be given in edema or when circulation is poor. Serious consequences are frequent when such treatment is given in cardiac failure.

As previously stated, Fischer's theory of edema in nephritis has not been generally accepted, and the treatment he advises, based on his theory, may often be dangerous.[100] Some support of his treatment and some suggested limitations have been offered by Lowenburg.[101]

During health, and in fact during life, very slight changes in the reaction of tissues occur. To keep the blood alkaline, or better, "neutral," according to Adler and Blake,[102] the bases "chiefly bicarbonate and alkaline phosphate, must equalize the acid substances at all times. The function of the bases of the blood is to transport acid, and to help to maintain the proper concentration." If more acid were introduced into the system than could be neutralized, acid intoxication would result. The stronger the acid chemically, the more readily it combines with the base. Hence in certain pathologic conditions stronger acid than carbonic acid may be produced and attach itself to all the available base, so that this is insufficient to carry carbonic acid from the tissues to the lungs. Many serious symptoms result, culminating in coma, due to acidosis. This occurs typically in diabetes when beta-oxybutyric and diacetic acids develop. As not all the ammonia in acidosis is converted into urea, some of it directly neutralizes the extra acid, and the amount of this ammonia combined with acids excreted into the urine is an index of the degree of acid intoxication.

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[100] Fischer, M. H.: The Treatment of Nephritis and Allied Conditions, THE JOURNAL A. M. A., May 31, 1913, p. 1682; Relation Between Chlorid Retention, Edema and "Acidosis," Jan. 23, 1915, p. 325.

[101] Lowenburg, H.: Efficiency of Sodium Chlorid in the Therapeutics of Bright's Diseases, THE JOURNAL A. M. A., Nov. 28, 1914, p. 1906.

[102] Adler, H. M., and Blake, Gerald: The Retention of Alkali by the Kidney with Special Reference to Acidosis, Arch. Int. Med., April, 1911, p. 479.

In edema caused by circulatory insufficiency the withholding of salt may not be beneficial, while the consumption of an added amount of salt, provided the ingestion of water is reduced to a minimum, may assist in removing the edema by increasing the water requirement of the blood. While in the edema of circulatory insufficiency the blood may thus be stimulated to pick up the transudates of liquid, in nephritic edema accompanied by inability of the kidneys to excrete salt, the addition of salt to the diet increases the edema; it must therefore be withheld in order that the circulation may gradually remove the exuded liquid. If the kidneys are able to throw off salt, no harm may be done by allowing the patient to have it, or by giving him more water.

The reason for diverse findings and reports concerning the advantages of a salt-free diet in edema is probably that in many instances there was circulatory disturbance and passive congestion of the kidney in which salt not only might not be detrimental, but in conjunction with digitalis might be of great benefit in removing the edema. However, in real nephritis with edema and high pressure, salt is injurious and water should be imbibed only in small quantities.

Physiologically, we need about 2 gm. of sodium chlorid daily to keep up the sodium chlorid equilibrium of the tissues. The greater portion of this substance is eliminated by the kidneys in from twenty-four to forty-eight hours, while the remainder is excreted by the skin, and intestines, and by tears. In most cases of real nephritis the salt excretion is retarded, and it may be greatly retarded. The rate of elimination may vary so that a period of retardation may be followed by a day or two of increased rapidity of excretion as the kidneys temporarily improve. Extensive retention of sodium chlorid is usually followed by edema, which lessens or disappears if the elimination of salt is again begun.

It has been shown that the bromin ion affinity is greater than the chlorin ion affinity; hence when bromids are administered during kidney insufficiency, the bromin may be excreted at the expense of the chlorin, the chlorids being retained. If the kidneys are seriously impaired, both chlorin and bromin may be retained.

It is self-evident that although edema in nephritis may not be entirely due to the inability to eliminate salt, a salt-free diet, for a short time at least, is distinctly advisable. If a salt-free diet is too long continued, the patient may suffer either from insufficient formation of hydrochloric acid in the stomach or from the lack of sodium chlorid in the tissues. Hence, after a period of salt-free diet, a small amount of sodium chlorid should be allowed daily, perhaps as much as 2 or 3 gm. Of course in nephritic edema the drinking of water should usually be restricted, even when the urine is scanty. When this precaution is taken, there will naturally be a loss in weight. A nephritic patient should be weighed frequently. If in spite of a limited diet and a limited ingestion of water the patient does not lose weight, an internal edema with retained sodium chlorid is the probable cause. Elimination of salt from the diet will reveal whether or not the ability of the kidneys to excrete salt may be improved; it may be that they will again excrete it in sufficient quantities and for a prolonged period, even in chronic nephritis. Bread should be made without salt; saltless butter should be used, as ordinary salted butter contains about 1 per cent. of sodium chlorid; fruits contain no salt; milk contains only a trace of salt; cheese is ordinarily salted, varying in amount up to nearly 3 per cent.; eggs contain only a trace of salt.

Magnus-Levy[103] states that muscle contains the least sodium chlorid of the body tissues, namely, 0.1 per cent. Connective tissue is the richest in sodium chlorid, and contains from 0.3 to 0.4 per cent. The entire body in a healthy man contains 130 gm. of salt, and the intake with an ordinary diet is from 15 to 20 gm. a day. If a normal person eliminates salt entirely from his diet, he generally loses weight.

Protein food requires more water in its metabolism and excretion than do other foodstuffs, but less food is consumed if water is not drunk with the meals. While a salt-free diet is most efficacious in diffuse nephritis with edema, it may also be of some value in edema of cardiac insufficiency, and in cirrhosis of the liver. Sometimes a large exudate, such as a pleuritic or peritoneal exudate, may be absorbed on a salt-free diet. Occasionally edema may not occur when sodium chlorid is retained.

NUTRITION IN NEPHRITIS

Diet in Acute Nephritis.—As already mentioned, a modified milk diet should be prescribed for the patient suffering with acute nephritis. The milk may be given hot or cold, preferably hot, sometimes predigested, sometimes diluted with a small amount of charged water, according to the patient's digestive ability. Fresh skimmed milk is frequently best for adults. As the phosphates are insufficiently excreted in nephritis, the phosphates of the milk may be precipitated into the intestine by precipitated calcium carbonate (from 0.5 to 1 gm.) taken three or four times a day. This, together with the phosphates of the milk, forms an insoluble compound, which is excreted by the intestine. Barley water is another beverage valuable for its nutritive properties, as a drink, and for its demulcent action on the gastro-intestinal tract. It may be advisable to give the patient well cooked and strained oatmeal gruel once or twice a day. Other simple, well prepared cereals may be substituted for the oatmeal.

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[103] Magnus-Levy, A.: Dehydration by Dietetic Measures, The Journal A. M. A., Dec. 17, 1910, p. 2139.

As water is difficult of excretion in acute nephritis, the amount allowed the patient should be limited, according to the amount of urine excreted and the extent of the edema.

The patient's nutrition may be well maintained, not only for many days, but even for several weeks if necessary, on as limited an amount as 60 gm. of protein a day, although as soon as the kidney excretion is sufficiently increased it is advisable to increase this allowance to 90 or 100 gm. a day. The retained nonprotein nitrogen of the blood indicates the advisability of increasing or decreasing the protein element. Two quarts of milk contain slightly more than 60 gm. of protein; but many patients cannot well digest that amount of milk, and if the nitrogenous waste is not excreted by the kidneys it will not be readily excreted by the intestine or with the help of profuse perspiration. However, unless uremia or entire suppression of the urine contraindicates this course, a diet of from

1 to 3 pints of milk a day, barley water being used to dilute the milk or as an additional drink, together with a thin oatmeal gruel once or twice a day, furnishes sufficient nourishment during the active period of acute nephritis or during an acute exacerbation in chronic nephritis. It may be advisable to add cream to the milk in the proportion of one-half pint of cream to 3 pints of milk, or it may be better to use skimmed milk, according to the patient's ability to digest milk, the amount of gastro-intestinal disturbance caused, the ability of the kidneys to function, and whether or not the patient is uremic.

When the volume of urine begins to increase in this disease, there is an added excretion of solids. In the later stages of chronic nephritis this is frequently not true. When the kidneys are recovering from acute inflammation, although albumin and casts may persist in the urine for some time, the diet may be gradually increased by adding more cereal, rice, potatoes, eggs and fruits which do not contain benzoic acid, such as oranges, lemons and apples. It is as well to omit meat from the diet as long as albuminuria continues. If this condition continues longer than two months, however, the case becomes one of subacute or chronic nephritis, and the diet should be arranged accordingly, meat being allowed in small quantities. As soon as convalescence is established in acute nephritis, the patient should be given small doses of iron daily, and certain fresh vegetables, including beans and peas, which are rich in protein, should be allowed. Toast or zwieback may be given early in the treatment, the amount being gradually increased during convalescence.

The stage in the disease at which the amount of water allowed the patient should be increased is a question for careful consideration. If after a number of days the edema has not disappeared and the kidneys excrete only a small amount of water, it may be well to try the effect of giving more liquid. If this results in an increase in urine, the patient's system requires the added water; if, on the other hand, the urine is not increased, the water intake should again be reduced to the minimum. However, when the kidneys are functioning well there is no advantage in forcing them to overwork by administering large amounts of water.

If the necessary treatment causes profuse perspiration, and if the impairment of kidney function causes the retention of poisons and nonprotein nitrogen in the blood, these poisons must become more concentrated. The same is true when it is deemed advisable to produce watery bowel movements. Consequently, the effect of profuse catharsis and of profuse sweating on the character of the blood must be considered, as uremia may be precipitated by too strenuous treatment. Therefore, when profuse sweating occurs, or if profuse catharsis is caused, plenty of water should be imbibed. Although many toxins and salts may be excreted by the bowels, only a small portion of the poisons retained by insufficient kidneys is excreted by the skin, which also carries off a small amount of salts, as evidenced by the staining of the clothing, the finding of crystals on the skin, and the odor of urine from the perspiration. Emphatically, however, it is most unlikely that profuse perspiration, or catharsis to prostration, is indicated or of advantage in acute nephritis or in uremia. A diet such as will reduce nitrogen waste and salts to the minimum, and, if necessary, venesection to relieve the system of a certain amount of toxin-carrying blood, appears more rational than the doubtful method of concentrating the poisons in the blood.

If uremia apparently is imminent, the diet should be reduced to the smallest amount, perhaps a pint of milk and a pint of barley water a day. If edema is extensive, the amount of water taken may be reduced to a tablespoonful or an ounce at a time. If the patient does not improve rapidly, the treatment becomes that of uremia.

When the elimination of salt from the diet has resulted in the absorption of the edema or effusions to a certain extent, small amounts of salt and a little more water may be added to the diet. If the patient continues to improve and the edema does not increase, the salt and water may be gradually increased to the normal amount. Too long abstinence from sodium chlorid is detrimental to the tissues, and interferes with the normal production of hydrochloric acid in the stomach.

When an acute congestion or a subacute inflammation of the kidneys develops on a chronic nephritis, the treatment becomes that of acute nephritis.

Diet in Chronic Nephritis.—In changing the diet of a patient suffering with chronic nephritis, it should be recognized that it is inadvisable, for the sake of assisting the kidneys to overwork or injure other parts of the body. Care must be taken not to deprive the patient too long of needed elements of nutrition; nor should his fuel be radically changed from anthracite to bituminous coal, so to speak, as this will result in a radical change in his digestion and metabolism. A temporary radical change in diet or a gradual change to a radically different diet may be advisable; but the effect of this procedure on the patient must be carefully noted.

The necessity of withholding salt from the diet has already been discussed. The amount of nitrogen allowed in chronic nephritis is also of great importance; frequently nitrogen may be excreted when the salt is not; but the excretion of both nitrogen and salt is generally impaired.

Frothingham and Smillie[104] find, as have others, that the phenolsulphonephthalein excretion is not much modified by diet. They find that the blood pressure is not much lowered in chronic nephritis by a low nitrogen diet, but it does fall on rest, and when a low salt diet has eliminated retained sodium chlorid.

When the nonprotein nitrogen of the blood is increased, a low nitrogen diet generally reduces it, so that apparently the only valid reason for giving a very low protein diet in chronic nephritis is the elimination of nitrogen from the blood. High protein diets in chronic nephritis should be entirely prohibited, although, unless the case is one of uremia, a sufficient number of calories of protein must be given to keep up the strength of the patient.

No disease requires more careful regulation of diet than chronic nephritis. Excess in any elements of food necessary for nutrition causes loss of appetite, indigestion and retention of waste matter.

Not more than 90 gm. — or 3,000 calories of protein, represented mostly by carbohydrates and fats — and salt and water in exactly sufficient quantities should compose the diet when elimination is fairly good with an ordinary amount of exercise. The greater the muscular exercise, provided the elimination is sufficient, the greater the amount of food required. Strenuous physical exercise may, however, cause an increase of nitrogenous waste (especially creatin), which impaired kidneys cannot rapidly eliminate, and poisoning, verging on uremia, may occur. Hence such exercise should be prohibited. Also exposure to severe cold should be avoided.

When nitrogen is retained, the amount of protein consumed must be reduced; in overweight the amount

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104. Frothingham, Channing, Jr., and Smillie, W. G.: A Study of Different Nitrogenous Diets in Chronic Nephritis, Arch. Int. Med., February, 1915, p. 204.

of starch and fat must be reduced; in renal edema the amount of salt must be reduced; in insufficiency of the heart the amount of water must be reduced. When estimating the amount of protein intake, it should be remembered that milk and skimmed milk are rich in proteins. A straight milk diet for a chronic nephritic is usually to be avoided, as quarts of milk must be imbibed in order to obtain the required number of calories of protein. The ingestion of this great quantity of milk causes indigestion, overtaxes the heart, may cause hydremia and edema, and places too great a strain on the kidneys. The caloric and protein value of foods need not be set forth here, as this information is accessible in books on diet.

The diet selected should be based on the excretory ability of the kidneys,[105] the nonprotein nitrogen content of the blood, the condition of the heart, the blood pressure, the state of the digestion, the weight of the patient, and the physical and mental work required of him. The elements of this carefully selected diet should be varied from day to day (though each week may represent the food of the week before), as otherwise the patient loses his appetite and food becomes repugnant to him. It is well for the patient to have nothing but skimmed milk one day each week. This allows his blood and tissues to be relieved of retained excretory products. Also, if possible, one day of complete rest each week is advisable.

It has not been shown that fresh fish, poultry and meat (not rich in purins, as kidneys, sweetbreads, liver and shad roe) are any more harmful to the nephritic patient than are vegetable proteins, such as nuts, peas, beans and oatmeal; but some patients may tolerate vegetable proteins better. There is no special difference between red and white meat, though the latter may be more easily digested. Boiled meats are better than those cooked in other ways because some of the extractives have been lost. For this reason meat soups should not be allowed to patients with nephritis, as they contain mostly meat extractives.

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[105] For test diets for nephritics see Janeway: Am. Jour. Med. Sc., 1916, **151**, 157.

With extensive edema, so-called diuretic drugs are inadvisable; although for a short time they stimulate the kidneys to a slightly greater activity, these organs soon tire and the excretion diminishes.

As a diet treatment the Karell milk cure, first suggested by Phillippe Karell, in 1865, is often successful in edema due to cardiovascular-renal disease, but is not as valuable in other types of dropsy.

The Karell treatment may be carried out as follows: The patient receives 200 c.c. of milk, in four doses, at four hour intervals during the day, beginning early in the morning. This treatment is continued for about a week. The milk may be taken hot or cold, as the patient desires, but he is allowed no other fluid and no solid food. After this period of limited diet he is allowed one egg and a slice of dry toast a day, for two or three days; the food is then gradually increased during the next two weeks by adding an egg, more bread, rice or other cereal, so that at the end of this period he is back to his ordinary diet, that is, the diet considered correct for one in his condition. Whether or not a little meat should be allowed depends on the character of the nephritis and on the absence or presence of uremic symptoms. During this starvation period an enema or some vegetable cathartic should be used. If the patient suffers from extreme thirst, the amount of water allowed may be slightly increased, or if hunger becomes unendurable, he may be allowed a little more toast. If the edema is largely cardiac, he may be allowed a small amount of salt.

This is a rigid form of the Karell treatment. Modification of this treatment is made by limiting the diet to skimmed milk and allowing more cereal.

Whether digitalis should be given depends entirely on whether the edema is cardiac or renal. If the chronic nephritis is apparently superimposed on a chronic heart insufficiency, digitalis may be of marked benefit; on the other hand, if there are symptoms and signs of uremia, and if the edema is largely due to renal insufficiency, digitalis may make the condition worse. When the blood pressure is high, the retention nitrogen increased, and the output of urinary solids greatly decreased, digitalis is most decidedly contraindicated.

When there is a sudden change in diet, such as a change from milk to meat or from meat to milk, the albumin in the urine may be found temporarily increased, probably returning, in a few days, to the normal amount.

The great danger from meat extractives, meat broths, etc., lies in the creatin present in these extractives, as it has been shown that when creatin and creatinin are increased in the blood the condition is menacing. Hence the patient with chronic nephritis should not be allowed to eat meat stock soups, meat broths, or large quantities of meat. Eggs and milk contain very little creatin, and vegetables not any. When meat is entirely removed from the diet, such creatinin as appears in the urine must be produced by the patient's own muscles.

However true it may be that alcohol does not often directly injure the kidney structure, as overindulgence in alcohol results in gastro-intestinal indigestion and disturbance of the functioning power of the liver, secondary irritation of the kidneys inevitably occurs.

Consequently, a person with chronic nephritis should, as a rule, be forbidden to partake of alcohol in any form.

In deciding the amount and character of the protein diet sufficient for a patient with chronic nephritis, we must not underestimate the needs of the body. If, on an ordinary protein diet it is found that the blood retains nonprotein nitrogen, the protein must, for a time at least, be reduced. If, as recently shown by Mosenthal and Richards,[106] even the consumption of large amounts of protein does not increase the nitrogen output or cause retained nonprotein nitrogen in the blood, the extra nitrogen or protein must be stored somewhere in the body, and we deduce the fact that this added protein was necessary to the patient. In other words, we must be careful not to underfeed the patient while trying not to overfeed him. There is no doubt that the life of many a nephritic patient has been shortened by the absorption of too little protein and too much milk, just as many a diabetic patient has suffered excessively from the lack of carbohydrate food.

TREATMENT OF CHRONIC NEPHRITIS

The therapy of chronic nephritis really involves the control of the disease and the management of the patient suffering with it. The title presupposes that we are not discussing an accidental albuminuria with or without casts, a lordotic (orthostatic) albuminuria, localized nephritis, or a kidney disturbance arising from a so-called surgical lesion, the cause of which must be treated before the management of the nephritis can be considered.

When the case has been diagnosed as one of chronic nephritis, its character and the probable prevailing type of lesion should be ascertained. If the nephritis has resulted from an acute infection, if it has become chronic in a young person, and if, judging from the amount of albumin, casts and edema, it is apparently a parenchymatous form of inflammation, the prognosis is unfavorable. If there are eye lesions, the prognosis is still more serious. If there is severe anemia, severe indigestion, and signs of chronic myocarditis, the prognosis is unsatisfactory. If the nephritis has apparently developed on a cardiac lesion, the prognosis may not be unfavorable, but a considerable although slow improvement may be expected. If the amount of urine is large and of low specific gravity, if there is only a limited amount of edema, if the patient is elderly, and, from the general heart tone, one concludes that the prevailing pathologic condition in the kidneys is of the interstitial variety, there is reasonable hope for a temporary cessation, or a retardation in the progress of the disease. True Bright's disease is cardiovascular-renal disease, the prognosis of which depends on the efficiency of the heart and the condition of the blood vessels. From the condition of the patient, may be surmised, but not positively diagnosed, whether this disease will eventually culminate in death resulting from uremia, angina pectoris, sudden cardiac dilatation, or rupture of a blood vessel in the brain.

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[106] Mosenthal, H. O., and Richards, A. E.: The Interpretation of a Positive Nitrogen Balance in Nephritis, Arch. Int. Med., February, 1916, p. 329.

All positive causes for the chronic nephritis must be investigated, the probable causes, if possible, removed, and anything in the patient's life or situation that would aggravate the condition should be changed. Physical strain, an injurious position of the body, accidental or maintained at work, a faulty bed, abdominal ptosis — everything which tends to interfere with the kidney circulation should be corrected. Chronic localized infections should be sought and removed, such as infections of the gallbladder, of a gum, of a tonsil or of a sinus connected with the upper air passages. It is possible that the urinary symptoms — whether albumin or casts — may be the remnants of an acute or subacute infection of the kidneys which has not yet cleared up; if this is probable, rest, careful diet and warmth, or a change of climate, may bring about entire recovery. Repeated tests of function will show the excretory ability of the kidneys, and may aid in establishing the cause of the nephritis.

All removable causes and irritants having been abolished, the treatment of chronic nephritis becomes (1) control of the diet, and (2) of the clothing; (3) care of the skin; (4) hydrotherapeutic measures; (5) exercise; (6) care of the bowels, and (7) necessary medication.

CONTROL OF THE DIET

1. Although the diet in nephritis has just been discussed, some repetition and elaboration is pardonable on account of its importance. It may be advisable in the form of chronic nephritis in which the parenchymatous apparently predominates to eliminate meat entirely from the diet, at least temporarily, and to reduce the protein intake to the least amount which will sustain strength and prevent anemia. Scientifically, repeated function tests, repeated study of the twenty-four hour urine excretion on a known diet, and the blood tests for nonprotein nitrogen will indicate the best diet for the patient. It should be such as will not cause gastro-intestinal indigestion. If meat is to be allowed, red meat may be eaten as well as white in small quantities once a day. It should be remembered that fish, chicken and fowl come under the heading of meat. All beef extracts, beef teas and meat soups should be prohibited. Rich nucleoproteins should be restricted, if not prohibited; this includes

liver, sweetbreads, shad roe, etc. If the blood pressure is not high, the patient may be allowed coffee and tea. On his digestion depends the amount of starch he may have; also whether the milk he drinks shall be skimmed or not. If meat is not allowed, the patient should eat more eggs, beans, peas, oatmeal gruel, bread, nuts and cheese.

The amount of water taken must be regulated according to the edema and the patient's need of water. If the specific gravity of the urine is low, as occurs in interstitial nephritis, he requires plenty of water to eliminate the salts. If edema is extensive, the amount of water and salt used must be reduced.

A patient on a meat-free diet should receive a small amount of iron daily. If this salt-free diet is long continued, he will probably need a small amount of dilute hydrochloric acid, not more than 5 drops, two or three times a day, after meals, according to the character of his food — a pure starch meal would not require hydrochloric acid.

The patient should be frequently weighed, and the hemoglobin reading frequently taken. If he is losing weight and edema is not reduced, the food is not sufficient. If the hemoglobin index is diminishing and there is actual loss of red corpuscles, his protein is insufficient, and he is not receiving, or at least metabolizing, a sufficient amount of iron. If the albumin excretion is large, he may lose weight and become anemic in spite of a proper diet. Such a patient should rest for a time. An increase in weight does not necessarily mean that the patient is improving, as this may indicate an increase of edema, perhaps of the concealed or internal variety.

Great fatigue on the least exertion may be an evidence that the patient's food is insufficient, that his heart is failing, or that he is toxic. Analysis of the blood pressure and the character of the heart action will show whether the heart is at fault or whether the blood pressure is too high and is causing dyspnea and fatigue. The hydrotherapeutic measures, sweating procedures, physical exercise or purging may be too severe, and should be modified. A blood test may show that he is retaining nitrogen. Under any circumstances, such fatigue, especially if associated with muscle cramps and generally increased nervousness, should be carefully investigated. Uremia may be impending.

Game meat should be avoided; fish and shellfish should not be eaten unless strictly fresh. While cheese is one of the best protein nutriments for a meatless diet, in certain instances it causes indigestion and should be avoided. All spices and highly seasoned sauces and dressings, canned foods and asparagus should be prohibited. Potatoes, turnips, parsnips, carrots, peas, beans, string beans, spinach, cabbage, cauliflower, watercress and celery may be eaten. In a meatless diet the protein nutriment is obtained from eggs, bread, milk, oatmeal, peas, beans and perhaps cheese. Nuts, thoroughly comminuted and swallowed as milky pulp, may be freely eaten if not difficult to digest. The amount of cereal, bread and potatoes which will be of profit to the system depends on the weight of the patient. He should not be allowed to gain weight, and surplus weight should be reduced if possible. When an obese person suffers from chronic nephritis, the most careful arrangement of diet and exercise is necessary to reduce the weight, to sustain the circulation, and to cause the kidneys the least possible irritation. If the patient is not obese, it is desirable that a moderate amount of sugar should be allowed. Fruits should usually be included in the diet unless contraindicated by the activity of the bowels and by the causing of gas or hyperacidity. Vinegar and pickles are usually injurious; on the other hand, dilute hydrochloric acid may at times be needed to aid digestion, as indicated by gastric indigestion and a heavily coated tongue.

In radically changing the ordinary diet of the patient with chronic nephritis, care must be exercised not to place too great a task on the various organs of the body. Many a patient who is suffering from chronic nephritis will thrive much better on a modified mixed diet, including meat, than on a heavy starch and vegetable protein diet.

The state of the blood pressure indicates whether or not the drinking of coffee and tea may be allowed. If the systolic blood pressure is above 160, or the diastolic above 105, all caffein should be avoided; otherwise an ordinary amount of tea and coffee need not be prohibited. Alcohol should be prohibited entirely, unless the patient is well advanced in years and has been accustomed to it, when a small amount may do no special harm. When there is evidence of congestion or disturbance of the liver, or when there is much intestinal indigestion, alcohol should always be prohibited.

A man accustomed to tobacco may use it moderately, unless the blood pressure is excessively high. In a habitual smoker who has stopped smoking, an irregular heart may be steadied by a return to the moderate use of tobacco. When an immoderate use of tobacco is forbidden a high blood pressure patient, the circulation should be studied and the amount of tobacco allowed found to be moderate in his particular case. If he can give it up entirely without circulatory debility, or if it can be reduced in amount gradually to total abstinence, without weakening the heart, this should be done.

THE CLOTHING

2. A nephritic patient should avoid being chilled, since, if his skin is cold or chilled, internal congestions occur. As nephritis progresses the skin usually becomes dry, normal perspiration decreases, and in the later stages may be almost absent; consequently, that which tends to promote the health of the skin as an excretory organ is advantageous. Though warm clothing should be worn during the cold season, this does not signify that constant profuse sweating should be caused. The patient should be covered warmly, at night, but should have plenty of fresh air to breathe.

If possible, the winter should be spent in a warm, dry climate, that of Arizona, New Mexico or the central part of Southern California being preferable; or he may go to a more humid warm climate, like that of southern Florida, the West Indies, or the Hawaiian Islands. As a general rule, a hot dry climate is better for chronic nephritis than a cold dry climate, but either is better than a changeable moist climate.

Daily warm baths are of great benefit to these patients. If in a cold climate, the bath should be taken just before retiring. Hot, stimulating baths are not advisable; cold baths should be entirely avoided, and sea bathing or fresh water bathing, at any season of the year, in whatever climate, should be prohibited.

3. The skin should be kept soft and pliable by warm bathing and by exercise, and if exercise is impossible, by massage. If the skin becomes dry, oil of theobroma (cacao butter) or other bland oils should be rubbed into it. When, notwithstanding these measures, the skin ceases to be a good excretory organ, prospect of recovery is discouraging.

The greater the insufficiency of the kidneys, and the greater the amount of salt thrown off by the skin, the more itching there is likely to be. This itching is not as serious a sign as in liver disturbances and in jaundice, but may cause scratching and consequent eczema. Anything promoting circulation in the skin, such as warm baths, gentle massage or the rubbing in of oil, tends to prevent or stop the itching. Any particular eczematous spot should be treated as any other eczema with a local sedative, or, if necessary, with stimulating ointments. The water used for bathing should be as soft as possible — bran may be used to soften it. The skin should be dried with soft towels.

4. Hydrotherapy promotes the normal excretion and health of the skin. Another indication for hydrotherapeutic measures is obesity and high blood pressure. The extent to which these methods should be employed depends entirely on the sufficiency of the circulation. If the heart is weak, even though the blood pressure may be high, only the mildest water treatments should be used. The Turkish bath is perhaps the most popular of the severer hydrotherapeutic measures. If the patient is obese, reacts well, perspires freely, and rests after the treatment, such a bath taken once a week is beneficial to him. He should be allowed to drink plenty of water after this bath so that the urine will not become too concentrated from the withdrawal of water through perspiration. Severe cold applications after such baths are inadvisable. Unless the kidneys are severely injured, the principal excretion by the skin is water, with a certain amount of sodium chlorid. Other salts are excreted only in minute quantities. If edema occurs, the only heat baths advisable are the electric light cabinet baths, or body baking, and the cabinet bath has really superseded the body bake, as exhausting the patient less and having a less injurious effect on the heart. These measures reduce the blood pressure, temporarily at least. Provided these measures are adapted to the circulation, they are beneficial

and relieve kidney congestion to a certain extent. These baths are not needed in warm climates. Care should be taken that the patient does not become chilled or catch cold afterward. The majority of patients, however, will derive as much benefit from warm baths taken at home, especially if followed by massage, and if the diet is suited to the patient's condition and weight. If a patient thus "knocked out," so to speak, by the severity of the treatment, is very thirsty, and drinks a considerable amount of water, or is very hungry and eats too much, and if the blood pressure immediately rises to its former height, the benefit derived is reduced to a minimum. These measures are most emphatically contraindicated in any patient with cardiac insufficiency, or who is prostrated by the treatment.

High frequency current treatments to reduce blood pressure are inadvisable for a patient with chronic nephritis. The blood pressure is reduced only temporarily, if at all, and the patient is often made nervous by these treatments. Other measures to reduce blood pressure are preferable.

The administration of radio-active waters for chronic nephritis is still so little understood that their value can neither be asserted nor refuted.

The application of hot packs and other local heating measures in chronic nephritis will be described under the treatment of uremia, as they are indicated only in uremic conditions.

Carbonic acid baths, whether taken at springs and cures, or under artificial conditions at home, should ordinarily not be used in chronic nephritis. It should also be remembered that hypertension is only one symptom of chronic nephritis, and whether or not such baths are valuable in the treatment of some forms of hypertension is not now under discussion.

5. The amount of exercise that a chronic nephritis patient should take depends on his circulation and weight. If he is overweight, and his circulation is good, considerable exercise should be taken, until good, healthy perspiration is produced, after which a warm bath and rub-down are advisable. If he is on the borderline of kidney insufficiency, overexertion may readily cause an imbalance, and any severe muscle strain or hard muscle work may precipitate uremia. The value of vigorous exercise used in a man's business or prescribed when his work is sedentary is to increase muscle nutrition and muscle circulation, to relieve the congestion of the organs of metabolism and excretion, and also to prevent the increase of fat. By such exercise, too, the circulation of the skin is increased and the skin excretions become more normal. If a patient has a weak heart or the blood pressure is high, exercise must be carefully regulated in order to aid and not injure him. The Oertel graded exercises should be modified to fit the individual. If he feels full-headed and has throbbing in the chest or cardiac pain and the blood pressure is increased, the exercise is excessive. If the blood pressure is too much lowered by exercise, it must be modified.

6. If the bowels do not move daily and thoroughly in chronic nephritis, they must be stimulated artificially. Daily catharsis is not necessary, and is harmful. If the patient is obese, a saline preparation taken daily before breakfast in sufficient amount to cause one or two movements may be the best treatment, depending on the amount of intestinal gas that it causes; if a large amount of gas is caused, a vegetable cathartic should be substituted. If the patient is not obese, the blood tension not excessively high, and the kidneys excreting fairly well, a daily vegetable laxative drug, as cascara, aloin, rhubarb or podophyllin, or any com-

bination of these drugs, is much better. Phenolphthalein should seldom be used in chronic nephritis. If the bowels can be regulated by the diet, that is, by the use of fruit, agar agar, whole wheat bread or bran bread, this is preferable, and no drug should be administered. Liquid petrolatum may be used for a time, if satisfactory, but when used very long it is liable to inhibit the secretion of the stomach and intestinal digestive juices, and to cause indigestion. It may make the excretions so oily that the sphincters do not act perfectly, much to the discomfort of the patient.

Once a week, unless the bowels move freely, catharsis may be caused by calomel and a purgative, by castor oil, or by a large dose of saline. The best time to take this is on the day on which the patient should ordinarily reduce his diet to the minimum, in other words, on a skimmed milk day. Retained nitrogen and salts are thus excreted by the bowels and kidneys, and the patient starts the week in a sanitary way, even though his kidneys are imperfect.

7. Besides laxatives and iron, when indicated, as little medication as possible should be given in chronic nephritis. The various measures suggested for the control of this disease will for a long time preclude the necessity for using drugs. If a hypnotic must be given for a short time, bromids or chloral should be selected on account of the usual high blood pressure. The synthetic hypnotics should ordinarily not be used, although an occasional dose of veronal-sodium or luminal may be employed. In high blood pressure nitroglycerin at night frequently induces sleep.

If the blood pressure is not kept down by physical methods, diet, etc., nitroglycerin, in a dose of $\frac{1}{200}$ to $\frac{1}{100}$ grain, three, four or five times a day, as found necessary, will almost invariably lower the blood pressure, unless the patient is uremic. If the patient is obese, especially if the skin is dry, the heart slow and the blood pressure high, 1, 2 or 3 grains of thyroid, given each morning, will prove beneficial, the dose depending on its effect on the patient. If it causes nervousness, sleeplessness or rapid heart, the use of the drug should be discontinued. Its purpose is to prevent the addition of weight, to lower the blood pressure, to stimulate the skin, and to aid in the detoxication of retained products of nitrogen metabolism. In nephritis occurring at the time of the menopause in women with signs of subsecretion of the thyroid, thyroid medication is very beneficial.

If the patient has no appetite, some simple bitter tonic may be given before meals; or, 1 grain of quinin sulphate may be given in capsule three times a day, with or without 1 grain of reduced iron.

If any coincident condition occurs in a patient with chronic nephritis, it must be treated as usual, with the exception of greater care in regard to the use of drugs. Any drug known to irritate or injure the kidneys should not be given. Iodids should ordinarily not be prescribed; arsenic usually is harmful; phenyl salicylate or any other salicylate is inadvisable. Codein may be used to alleviate pain, and morphin if the pain is severe; but one must always be extremely careful in the use of narcotics in chronic nephritis. Coal-tar products should be given only in very small doses. Digitalis, if used, should be given in small doses and with great care. In cardiac insufficiency it may be of great benefit; but if the kidneys are much impaired it may do great harm. Caffein in any form may act as a temporary diuretic, but it is liable to cause, secondarily, diminished secretion of the kidneys. It should not be given in high blood pressure, but may be used in acute cardiac failure.

The treatment of cardiovascular-renal disease has already been described.[107]

One is seldom justified in giving a so-called diuretic in chronic nephritis. It is inadvisable to stimulate an impaired organ, but if alkalies are indicated, potassium citrate may increase the output of urine, temporarily at least. Theobromin sodium salicylate is one of the least irritating of the caffein group, but often proves unsatisfactory in reducing edema or in increasing the output of urine.

Christian[108] has shown that theocin has no constant diuretic action. It may or may not cause slight diuresis when a patient has no edema. It generally causes diuresis in cardiovascular-renal disease with edema, and also increases the sodium chlorid excretion. The nitrogen execretion is much less increased, for even when diuresis is caused, Christian finds that the kidneys may later show a decrease in nitrogen excretion, or a kidney tire; therefore, the only condition in which to use theocin, one of the strongest of the newer so-called diuretics, is in cardiovascular-renal dropsy, when it should be used only intermittently and perhaps in conjunction with digitalis.

Large doses of thyroid extract may prevent convulsions and may aid in saving the life of a patient suffering with uremia from acute nephritis or with puerperal eclampsia.

There is no doubt that in the majority of cases the proper management of chronic nephritis will prolong the patient's life by months, and perhaps years. Partial uremic attacks may occur during the course of chronic nephritis, and by proper measures the patient's life may be saved. Some patients survive several attacks, but sooner or later a fatal uremia will occur, unless the patient dies of heart failure, apoplexy or some other disease or complication of diseases. The prognosis of uremia occurring in chronic nephritis, if the attack is severe, is less favorable than that of uremia occurring in acute nephritis. The treatment is that described under uremia. The value of decapsulation and the indication for it in chronic nephritis will be described under that heading.

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[107] Cardiovascular-Renal Disease, Therapeutics, The Journal A. M. A., Sept. 23, 1916, p. 951.

[108] Christian, Henry A.: Some Studies of a Diuretic (Theocin), Arch. Int. Med., November, 1916, p. 606.

Although the urine of a chronic nephritis patient should occasionally be analyzed, it should not be analyzed so frequently as to cause continuous mental worry. There is no reason why the chronic nephritis patient, like the patient with chronic heart weakness, should not live for years, if properly watched; but, as in heart disease, it is necessary to divert the patient's attention from himself, while we are giving him a more or less frequent supervision (depending on his condition).

UREMIA

The symptoms of uremia are the same, whether it is caused by acute or chronic nephritis. Ordinarily, when uremia is imminent, premonitory symptoms occur, such as severe headache, fulness of the head, dizziness and eye blurs, or "turning black before the eyes," as the patient describes it; there may be muscle twitchings, muscle cramps, restlessness, sleeplessness or drowsiness; there is often nausea, vomiting, and diarrhea—Nature's method of improving the condition. The blood pressure increases, and may be very high. The urine shows a decreased output of solids, especially of nitrogen, and the blood shows retained nonprotein nitrogen. The Babinski sign, as first pointed out by Curschmann,[109] is a prognostic sign of impending uremia, and probably occurs from the irritation of toxins on the cerebral centers. It is probable that in all cases of uremia, disturbances of the liver function play some active part. The liver forms urea out of ammonium salts. Exclusion of the liver in animals prevents the formation of urea, and death soon occurs, generally preceded by convulsions. When the liver function is disturbed by any disease, the poisons resulting from metabolism are not well detoxicated. Urea may be diminished in amount, and it has been suggested that ammonium carbonate remains in the blood, and that ammonium salts are irritant to the central nervous system. If the organic sulphates and indican are increased in the urine there may be an abnormal amount of substances circulating in the blood which are irritant to the central nervous system and which sooner or later cause disease.

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[109] Curschmann, H.: München. med. Wchnschr., Sept. 26, 1911: abstr., The Journal A. M. A., Nov. 4, 1911, p. 1576.

It may also be well here to caution against the use of the urea test of kidney function in conditions in which uremia is feared. Twenty gm. of urea, which is the dose for a urea test, should normally be eliminated in one and one half days. In nephritis the excretion may be greatly delayed or the whole amount may be retained. Hewlett, Gilbert and Wickett[110] have recently shown that in normal persons the administration of considerable amounts of urea by the mouth may greatly increase the urea content of the blood and cause such symptoms as "headache, dizziness, apathy, drowsiness, weakness and fatigue," the very symptoms which are premonitory of uremia. This shows how little extra nitrogen waste will cause imbalance of nitrogen metabolism in nephritis, and is the reason that chilling, extra muscular exercise, a high protein meal, or nervous and mental excitation, such as may occur during an after-dinner speech, a lecture, or after a controversy, may precipitate a uremic attack. This fact is daily verified by newspaper reports concerning prominent public men. An attack may be precipitated apparently out of a clear sky, without forewarning edema or other symptoms of kidney insufficiency. Such precipitate uremia usually occurs with the so-called interstitial variety of nephritis, and is often fatal.

Uremia may occur in chronic nephritis when the pathologic condition is largely interstitial, without the presence of albumin and casts in the urine. Mild uremic attacks may not be fatal in chronic nephritis; all the symptoms of uremia may cease, and the patient may long be free from them, although ordinarily they

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110. Hewlett, A. W.; Gilbert, Q. O., and Wickett, A. D.: The Toxic Effects of Urea on Normal Individuals, Arch. Int. Med., November, 1916, p. 636.

will again recur until the fatal issue. In acute nephritis, even a severe attack of uremia with convulsions and coma may not be fatal, and the patient may completely and entirely recover. The prognosis, however, is always serious and generally dire; but one should never give up hope of saving the patient. However, when chronic nephritis has long been present and the patient has had repeated slight attacks of uremic symptoms, when true uremia finally develops the outcome is usually fatal.

A recent investigation by Chace and Myers[111] shows that increase of the uric acid in the blood is suggestive of incipient nephritis; that changes in the amount of urea in the blood in nephritis is a dependable indication of the condition of the patient, and that a large increase in the creatinin of the blood is of serious import, more than 5 mg. of creatinin in 100 c.c. of blood indicating an early fatal outcome, the creatinin content of the blood increasing rapidly just before death. Morse[112] has shown that the creatin is normally greatly increased in the urine after childbirth.

The symptoms indicative of uremia are: hypertension (a diastolic pressure of 120 is usually indicative of a serious kidney condition); headache (generally occipital or frontal); restlessness, or, sometimes apathy and somnolence; ringing of the ears; eye blurs; muscle twitching; cramps; coated tongue; nausea; vomiting; dry skin; odor of urine from perspiration and skin; dyspnea; finally, convulsions and coma.

The intensity of the foregoing symptoms varies, of course, with the degree of uremic poisoning, and the condition may frequently be aborted by proper treat-

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111. Chace, A. F., and Myers, V. C.: The Value of Recent Laboratory Tests in the Diagnosis and Treatment of Nephritis, The Journal A. M. A., Sept. 23, 1916, p. 929.

112. Morse, Arthur: Creatin and Creatinin Excretion During the Puerperium and Their Relation to the Involution of the Uterus, The Journal A. M. A., Nov. 6 1915, p. 1613.

ment in its incipiency. When several of these symptoms develop in nephritis, active treatment must immediately be instituted. A diminished output of urine usually accompanies these symptoms, although this may not always be true in contracted kidney. The pulse may be slow, becoming rapid, as the severity of the symptoms increases, unless considerable cerebral pressure causes its retardation for a time. If the condition becomes serious, the pulse may become rapid and weak, and the blood pressure lower.

Examination of the blood usually reveals an increase of nonprotein nitrogen in nephritis as uremia develops, but it should be noted that this increase has also been found in cholera and typhus fever. Foster[113] found that uremia and death may occur when the nonprotein nitrogen of the blood is not increased.

The exudates in nephritis have been shown to contain the same amounts of nonprotein nitrogen as the blood; hence, when in uremic patients an increased amount of water is lost by the bowels, by the kidneys or by perspiration, these fluid exudates are apt to be absorbed, and serious uremic symptoms may again appear as a result of the increased nonprotein nitrogen and other toxins in the blood. When uremia and finally coma occur without convulsions the amount of retained nitrogen in the blood has probably not been greatly increased since the more retained toxins there are in the blood, the more probable is the occurrence of convulsions.

Foster found that ammonia-nitrogen may or may not be increased in uremia, but that it is usually increased in convulsive cases and in those cases with a high total nonprotein nitrogen. Normally there is less than 0.5 mg. of ammonia-nitrogen in 100 c.c. of blood. The greatest increase of ammonia-nitrogen in the blood is found in diabetics and in diabetic coma.

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[113] Foster, N. B.: Uremia, Arch. Int. Med., March, 1915, p. 356.

Folin[114] found that the uric acid of the blood is increased in renal disease, while Foster found that the purin nitrogen is increased in nephritis, most markedly in those who have contracted kidney; in uremia it is twice the normal amount.

Corroborative of the findings of Foster that occasionally uremia may occur without an increase in non-protein nitrogen in the blood, Strauss[115] terms a condition "pseudo-uremia" in which the nitrogen retention is not great and the blood tension may not be high. These patients have a tendency toward convulsions and coma; the condition is closely allied to puerperal eclampsia. Convulsions, occurring when the kidneys are not apparently seriously insufficient and the non-protein nitrogen of the blood is not increased, point toward acidosis, and future investigations may show that the ammonia-nitrogen of the blood is increased.

TREATMENT OF UREMIA

The indications for the treatment of uremia may thus be itemized: (1) the diet; (2) elimination; (3) rest; (4) pain; (5) care of the circulation; (6) care of the central nervous system; (7) control of edema.

1. At the first evidence of uremia, the diet should be diminished to the smallest amount possible, such as a pint of milk a day. The use of water should not necessarily be restricted, unless edema is extensive and edema of the lungs is feared; if this is the case, it is well to restrict the intake of water in the endeavor to have the exuded liquid resorbed into the blood vessels and excreted in some way, if not by the kidneys, by the skin or bowels, as a certain amount of purging must be caused, and as perspiration, if not too profuse, is desirable. If, on the other hand, edema is not

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[114] Folin: Jour. Biol. Chem., 1914, **17**, 489.
[115] Strauss: Berl. klin. Wchnschr., April 12, 1915.

extensive, and catharsis and perspiration are caused, a liberal amount of water should be given the patient in order to facilitate these processes. It should be emphasized, however, that profuse catharsis causing circulatory weakness, or profuse perspiration causing exhaustion do not cure patients with uremia; furthermore, if water is not supplied and well absorbed into the blood vessels, the excretion of water from the blood vessels by the bowels or skin, or both, causes a concentration of the toxins which cause uremia. Hence, the nervous system must be more seriously irritated.

It may be well to note here, however, that Haldane and Priestley[116] have recently found that when large amounts of water are given to a normal person, and the output of urine is increased and the specific gravity lowered, the blood concentration remains the same. This was also true when profuse perspiration was caused. They came to the conclusion that a diminished hemoglobin percentage or an increased volume of blood was not caused by the intake of such amounts of water as would cause diuresis. This does not prove that in abnormal conditions, such as uremia, the blood may not become more concentrated by profuse diarrhea and sweating, or become less concentrated by the ingestion of sufficient amounts of water.

The scanty diet of a pint of milk or a quart of barley water a day, suggested above, cannot be continued more than a few days without the patient becoming seriously weak. If he is not comatose, the diet should be increased by some thin liquid cereal, with more or less alkali, such as sodium bicarbonate. The object of the alkali and the carbohydrate is to prevent the added element of acidosis. The alkali, however, should not be given in large quantities if there is serious edema, as it may aggravate this condition.

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[116] Haldane, J. S., and Priestley, J. G.: Jour. Physiol., 1916, **50**, 296, 304.

If convulsions seem imminent, the only food the patient requires is water, barley water, oatmeal gruel, malted milk, orange juice or lemonade, according to the condition of his central nervous system and stomach. If he is vomiting, and emptying his stomach well, hot water is the required treatment. If he is nauseated, but does not vomit, the stomach tube may be used to wash out the stomach, which will relieve him greatly. Occasionally bismuth and soda will soothe the stomach; a mustard paste over the epigrastrium may stop the nausea by counterirritation; or an electric pad may be of advantage. If more than a day or two is passed during which he is unable to retain nourishment, colon injections of water containing sodium bicarbonate should be given, as starvation may soon cause an acidosis which will add to the intoxication.

2. Bowel movement should be induced by compound jalap powder, 2 gm. (30 grains), repeated in four hours if free movements are not caused. Calomel or any other mercury may be injurious when a patient is uremic. If he is comatose and free bowel movements are urgently needed, 2 or 3 drops of croton oil on granulated sugar may be placed on the tongue, or may be given diluted, if the patient is able to swallow. If the bowels have moved freely several times, the patient should not be given another cathartic for twenty-four or thirty-six hours; but when the bowels have not moved for twelve hours or more, washing out of the colon with warm water is efficacious.

Profuse dripping perspirations are not essential in uremia. Great benefit apparently has been derived when hot air directed under the bed-clothing, actual body baking, steam bath (or some other procedure), has caused profuse perspiration. When patients survive such treatment, it is because enough dilatation of the blood vessels of the surface of the body has occurred to relieve the kidney congestion. But if the kidneys cannot so react, the blood is depleted of water, the retained toxins are in more concentrated form, the kidneys may not improve, and the danger is increased. Too profuse perspiration and general surface relaxation may also lower the blood pressure below the point desired, and the patient may die of circulatory failure. Hot packs to the back over the kidney region will cause enough local heat to be of more or less benefit to the kidneys by relieving local congestion, at the same time causing enough general perspiration to accomplish a relaxation of the skin without the use of more strenuous measures.

Pilocarpin is not usually indicated. It produces not only profuse perspiration, but also profuse bronchial secretion, so that the patient almost drowns in his own bronchial fluids. Frequently this flow of perspiration and bronchorrhea must be checked by atropin, if it is not too late. Drugs which act as strenuously as pilocarpin are not advisable.

3. Rest of mind and body is essential in uremia. The greater the muscle movements, the greater the nitrogenous waste; the more restless the brain, the more restless the body. When muscle cramps occur and convulsions seem imminent, a quieting dose of chloral, sufficiently large to be effective, should be administered, unless the patient is afflicted with cardiac weakness and low blood pressure. If the patient's heart is weak, if he cannot sleep and is very restless, codein should be given instead of chloral.

4. Some patients who are uremic suffer acute pain, either cardiac or abdominal; this acute pain must be stopped by codein or morphin, even though the impending coma may be hastened thereby. It is imperative

that agonizing pain be terminated, even in uremia. Fortunately, not many patients who are uremic suffer such pain; they are more likely to be stupid.

5. The blood pressure is generally high, and large doses of nitroglycerin are advisable; or the chloral resorted to for sleep, or the bromid used to soothe general restlessness will reduce the blood pressure. Catharsis and sweating also tend to lower the blood pressure. However, one must be cautious lest the suddenly lowered blood pressure be coincident with heart failure. If the heart is failing, the diastolic pressure becomes high, the systolic pressure is lowered, and the pulse pressure is shortened. This frequently occurs in the uremic condition of cardiovascular-renal disease, but whether or not uremia is present in that disease, digitalis, or perhaps an intravenous dose of strophanthin, should be given. However, it should be remembered that strophanthin may have too great an effect on the heart, if digitalis has previously been given.

If the blood pressure is very high, and apoplexy or sudden dilatation of the heart threatens, venesection should be done. This is also perhaps the most valuable treatment for convulsions or coma. The amount of blood withdrawn depends on the individual case. Subsequent to the venesection a colon injection should be given, the water containing a small amount of sodium bicarbonate, which should be allowed to be absorbed. If there is no edema or a very small amount, as in chronic interstitial nephritis, physiologic sodium chlorid solution may be introduced into the colon, the object being to fill the blood vessels again and to dilute the toxins they contain, thus reducing the irritation of the brain. Whether or not venesection has been done in uremia, colon irrigation is often of great value, with the water at a temperature of 110 F. A rectal tube may be passed a short distance, and, with the hips elevated, the water allowed to flow into the colon, where it may be allowed to remain a short time, and then siphoned out. This procedure should be repeated every three hours until several treatments have been given, as such irrigation tends to relieve the acute kidney congestion.

6. With frequent convulsions, chloral or bromids may be given by the mouth, or, if they cannot be swallowed, by the rectum; but the latter method of administration is not very satisfactory. Venesection, fomentations over the kidneys, heat, and croton oil catharsis are preferable. If frequent convulsions seem to threaten the life of the patient, chloroform inhalations should be given. Coma may be due to the intoxication, to cerebral pressure from an exudate into the ventricles, or from actual edema of the cerebral tissues. Spinal puncture will often drain fluid from the ventricles, but it would not be of benefit in edema of the cerebral tissues. Hot water bags to the feet and legs and heat over the abdomen will draw the blood from the head, and will lessen the cerebral congestion. Even with convulsions and coma, unless it is the terminal stage of a chronic nephritis, the patient may recover.

There is no detoxicant in uremic poisoning more successful than large doses of thyroid, and if 10 or 15 grains of this substance can be absorbed from the stomach there is generally an improvement, although it may be temporary. When convulsions seem imminent, and when uremic symptoms appear, thyroid should be given in 5-grain doses, or, with the stronger preparations now offered, in 2-grain doses, two or three times daily.

7. If the edema is cardiac, the treatment is that of cardiovascular-renal disease, and if the patient is uremic, digitalis or strophanthin may be of great value.

The blood pressure may not be high in a serious general anasarca, and caffein may be of value. Exhausting purging of the bowels should not be caused, as heart failure may result.

It may be necessary to remove large exudates from the pleural cavities or from the peritoneal cavity, but when such complications occur with uremic symptoms, the prognosis is almost hopeless. With such general anasarca, theocin may be tried; but it has been proved that this does not increase the output of nitrogen, although it may increase the output of sodium chlorid and water.

If the patient survives the uremic condition, and the kidneys begin to excrete, the treatment is that outlined for the convalescence of acute nephritis.

DECAPSULATION.

Decapsulation was first suggested by Edebohls, in 1901, and was done by him and by others with the hope that a new blood supply could be created around the kidney structure. Also, if the kidney was so acutely congested and swollen that the capsule formed a constriction, the release of this constriction might improve and save the kidney, or both kidneys, and, temporarily at least, prevent uremia and save life. This operation has not proved very successful, and certainly should not be done in chronic nephritis. However, decapsulation may be considered (1) in acute eclampsia when emptying of the uterus does not stop the convulsions; (2) in toxic nephritis (mercury, arsenic and phenol poisoning); and (3) in acute nephritis following an acute infection when uremia is present and there is complete anuria.

PUERPERAL ECLAMPSIA

If possible, this condition should be forestalled. Albuminuria, casts, and symptoms of kidney disturbance during pregnancy should, ordinarily, call for a termination of the pregnancy. Too great conservatism is not justifiable. The longer the kidneys are congested from the abdominal pressure, especially if nephritis has previously occurred, the greater is the hazard to the mother if an attempt is made to save the child, therefore such an attempt should not be made. If the kidney inflammation is not discovered until a short time before term, consultation may decide that it is best not to hasten the birth of the child. On the other hand, every day increases the danger, as the muscular efforts of the mother at the time of parturition load the blood with nitrogenous toxins, especially creatin, and eclampsia is almost inevitable.

If kidney defects are not discovered until parturition has begun, eclampsia may develop rapidly. In this case chloroform must be administered and the evacuation of the uterus hastened. Considerable hemorrhage from the uterus is conservative and life-saving, as the loss of a large amount of blood loaded with toxins may save the patient, after the manner of a venesection; such loss of blood is apparently even more effective in nephritis than that caused by venesection.

Of course it is recognized that puerperal eclampsia may occur when the urine has shown no signs whatever of nephritis. Thyroid in large doses is especially effective in this condition, and should always be given in puerperal eclampsia. After the uterus is evacuated, the subsequent treatment is that of uremia.

PATHOLOGIC CONDITIONS AND ANOMALIES

MOVABLE KIDNEY

A loosened kidney, generally without manifesting symptoms, may be found in about 20 per cent. of all women over 20. It is of infrequent occurrence in colored women, according to Griffith.[117] The right kidney is the one more frequently prolapsed. Griffith believes this kidney is found movable three times as frequently as the left. Occasionally both kidneys are prolapsed. Tumors of the kidney, or in the tissues above or around it, are perhaps accidental factors in causing a kidney to prolapse. Constipation is probably never a cause. Sudden loss of weight, that is, loss of abdominal fat, is perhaps a frequent factor, but it is usually associated with a predisposition to kidney displacement.

Griffith believes that a contracted lower part of the chest in women is an important predisposing cause, as such an anatomic condition makes the fossa that holds the kidney and suprarenal capsules shallow, and predisposes toward prolapse of the kidney. In men these fossae are generally deeper.

The kidneys are normally slightly movable, and the right kidney is lower than the left. When the fat, which is such an important factor in supporting the kidneys, is lost and one or both kidneys become abnormally movable, the slightest added strain will seriously displace one or both kidneys. When the corset is loose below and tight above, or when it is entirely removed and excessive dancing indulged in, as is now in vogue, loosened kidneys probably occur more frequently.

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[117] Griffith: Johns Hopkins Hosp. Bull., March, 1910, p. 75.

Werelius[118] quotes Glénard's classification of the degrees of prolapse of the kidney. In the first degree the upper end of a kidney is found at the costal margin; in the second degree the whole kidney is below the costal cartilages, and in the third degree the kidney is found anywhere in the abdomen at a distance from the costal cartilages.

The prolapsed kidney may be of any size, but it is often enlarged, and may become hydronephrotic. Various anatomic and histologic disturbances may be found in these kidneys, which are subjected to great variability in their blood supply, in their nutrition, and in the pressure of the urine in the kidney pelvis.

Secondary abdominal conditions caused by pressure from a floating kidney may occur, or there may be no symptoms, even though the kidney is very movable. Constipation is often a symptom, perhaps because of pressure on the colon or cecum.

Not infrequently chronic appendicitis is an accompaniment of a floating right kidney, and even ovarian pain and bladder irritability may follow. Gallbladder disturbances are sometimes associated with displaced right kidney. Whether these conditions are caused by direct pressure, by reflex disturbances, or by interference with the normal circulation of these parts is a question to be decided in each individual case. Both gastric and intestinal indigestion often occur in patients who have a displaced kidney, and there is frequently distress at night, and inability to lie on the side or on the back, without aches and pains. In many instances the displaced kidney is associated with ptosis of the stomach and intestine. Various neurasthenic and hysterical symptoms may occur in these patients, and if the loose kidney is not diagnosed, the patient may be treated at home or in sanatoriums for nervous dyspepsia, nervous indigestion, or similar complaints, with no permanent benefit.

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[118] Werelius, Axel: Nephroptosis and Nephropexy, The Journal A. M. A., March 1, 1913, p. 643.

It must be emphasized, however, that frequently floating and loose kidneys cause no symptoms whatever. When, during a physical examination, a displaced kidney is discovered, information of such a condition should, as a rule, be withheld from the patient.

Real pain, and many kinds of referred abdominal pain — even the intense pain termed Dietl's crisis — may be caused by a loose kidney. Dietl's crisis is really a kidney colic due to twisting of the ureter and distention of the pelvis of the kidney. Whether or not the obstruction is severe enough to cause one of these typical crises, the variations in the amount of urine retained in the pelvis of the kidney frequently cause the kidney to become enlarged. Hence many times scientific examinations alone can determine whether a kidney tumor has caused a movable, enlarged kidney or whether the kidney contains a stone with subsequent enlargement.

Other movable tumors may also be found in an abdomen, but the kidney shape is typical, and a loosened kidney is more movable than other abdominal tumors. Stone in the kidney and kidney tumors are apt to cause hematuria; hydronephrosis caused by the mobility of a kidney is generally intermittent; at one time a large tumor is found, at another time the kidney may even be difficult to locate.

It should be emphasized that while a single positive finding of a loosened kidney is diagnostic, repeated abdominal examinations may not reveal a loosened kidney, though a later examination may show that one is even floating. The elusiveness of such a kidney is probably caused by varying gas pressure in the intestine.

A movable kidney that is causing symptoms may be kept in place by a properly made and properly fitting corset, or by a so-called kidney belt. Rest and an increase in such a diet as will cause the patient to gain weight may cause such a kidney, if not actually floating, to become more or less permanently fixed. Such simple treatment as an abdominal belt sometimes causes entire abeyance of all disagreeable abdominal symptoms, and the patient is symptomatically cured. At times it is difficult to determine how many of the abdominal disturbances and functional disturbances are due to the displaced kidney, but strapping of the abdomen will generally show what value an abdominal belt or corset will produce. Hence, before an expensive apparatus is ordered, strapping should be tried, as the abdominal condition may not be caused by the loose kidney, which may be the source of no symptoms whatever.

The general trend of surgical opinion is that the importance of a loosened kidney in causing symptoms has been overestimated, that many patients are not improved by an operation to fasten the kidney to its normal position, and that many such plastic operations are unsuccessful, as the kidney again becomes loose. On the other hand, while a loose kidney does not necessarily need medical or surgical attention, there are many instances in which a belt does not ameliorate the symptoms, and in which an operation for fastening the kidney into its normal position will cause permanent disappearance of all associated abdominal disturbances. Therefore, the least that can be said is that occasionally such a plastic operation is necessary and is curative.

HYDRONEPHROSIS

This condition — a dilatation of the renal pelvis — can occur only as the result of some obstruction in the ureter. Such an obstruction may be due to an impacted calculus, a coagulum of blood, congealed pus, some tumor growth pressing on the ureter, a twist of the ureter caused by looseness of the kidney, or to long continued distention of the bladder. An obstructed ureter will rarely be found as a congenital condition. The longer such a dilatation of the pelvis of the kidney continues, or the more frequently such a condition occurs, the greater the injury to the kidney structure. Frequently, if the pressure of urine in the kidney becomes sufficiently great, it will either straighten a twisted ureter or dilate the ureter around an obstruction, and the kidney will empty itself into the bladder. If the calculus or other obstructive substance plugs the orifice of the ureter and is soon displaced, dilatation of the pelvis may not occur and such plugging may not recur although the calculus may still remain in the pelvis of the kidney. Any sudden obstruction or a twist of the ureter does not always lead to dilatation of the kidney pelvis, as the associated kidney congestion and a diminution of the excretion of urine sometimes prevents this. Repeated obstruction, however, will generally cause a hydronephrosis.

Anything that causes prolonged distention of the bladder, whether due to an enlarged prostate or to some urethral obstruction, may cause a hydronephrosis of one or both kidneys. A readily movable kidney may have a more or less dilated pelvis, even when Dietl's crises do not occur, although in this condition alone real hydronephrosis does not often occur. A gradual dilatation of the pelvis of the kidney may cause an abdominal discomfort from the weight and pressure on the adjacent organs, but perhaps no real pain. The polyuria following a Dietl's crisis is due not only to the outflow of retained urine in the pelvis of the kidney, but also to an increased secretion from the irritated kidney.

Hemorrhage into the pelvis may cause its dilation and a subsequent distention from plugging of the ureter. Such blood may come from a tumor, or from varices in the pelvis or calices of the kidney.

When the flow of urine is suddenly obstructed, shock may occur, as in renal colic, and the temperature may rise, as it usually does when the obstruction is caused by a plug of pus from a pyelitis or from a coagulum from a bleeding kidney. The more serious the cause of the obstruction, the more frequent become the attacks of acute hydronephrosis, pain, etc.

Urinary signs ordinarily do not occur when external pressure or a twisted ureter is the cause. If there are renal calculi, if the kidney is bleeding, or if there is pyelitis, the urine will show blood, or pus, or both.

There may be no absolute obstruction to the flow of urine, and yet the conditions may cause sufficient damming back to produce a dilated pelvis. This condition is discovered by cystoscopy, the passage of the ureteral catheter and by ascertaining how much fluid the pelvis will contain. Long continued irritation of a ureter may cause sufficient narrowing to prevent the passage of a ureteral catheter; or a constricted ureter may be congenital.

The sensation of pain when fluid is injected into the pelvis of the kidney will indicate when the pelvis is full. Although there is a difference of opinion as to the amount of liquid a normal pelvis should contain, it is probable that anything over an ounce (30 c.c.) should be considered abnormal.

While a large hydronephrosis may ordinarily be diagnosed by physical signs and symptoms, pyelography, the injection into the pelvis of the kidney of a solution that will give definite roentgenographic pictures, will positively determine the size of a kidney pelvis, and the relationship of an abdominal tumor to the kidney.

That any obstruction which causes a hydronephrosis may seriously impair the kidney structure has been shown experimentally, and Keith and Snowden[119] found that experimental hydronephrosis soon caused albuminuria, decreased phenolsulphonephthalein and lactose excretion, and increased the nonprotein nitrogen of the blood, causing renal insufficiency. Death may result from this condition, unless the other kidney is in perfect health. It should also be recognized that a hydronephrotic kidney may readily become infected.

The kidney probably becomes painful only when its pelvis is irritated, or when there is an irritation near its capsule. Consequently, disturbances located in the secreting kidney substance are rarely painful. One of the most frequent causes of kidney pain is distention of the pelvis perhaps by a calculus. Just how much distention the pelvis may tolerate without pain may be variable, but as previously stated, more than an ounce of fluid, sometimes less, will cause kidney ache, if not actual kidney pain. The patient may get relief at night from kidney pain due to pelvic distention or pelvic irritation only by lying on the affected side, so that the respiration will not cause as much movement of this kidney. Kidney pain and distress, accompanied by a diminished amount of urine, followed by the passage of a large amount of urine with relief from distress, points strongly to obstruction of the ureter and a dilatation of the kidney pelvis.

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[119] Keith, N. M., and Snowden, R. R.: Functional Changes in Experimental Hydronephrosis, Arch. Int. Med., February, 1915, p. 239.

At first such pain is referred to the lumbar region, and a tender point is discovered between the last rib and the erector spinae muscles, the point of tenderness being about the size of the end of the thumb. This pain is stabbing and severe, and may cause vomiting. As the kidney pelvis distends more and more, there is more general abdominal discomfort, and the pain and ache is diffused over the whole side and lumbar region. As the pelvis reaches its full capacity, distress is referred to the abdomen, more, of course, on the affected side, and the symptoms become more like those of renal colic; but the acute obstruction is likely to be shorter than that of a renal colic as the fluid forces its way downward. The pain is increased on standing up by the drag of the heavy kidney, and is lessened on lying down. The pain may be as agonizing in Dietl's crisis as a renal colic. The pain, if right sided, may be referred to the gallbladder region, to the appendix or to the pelvis, and render diagnosis difficult, at least while the pain is acute. But a tumor in the kidney region, or a movable tumor, with the history of the case, will generally prevent an unnecessary immediate operation. In a doubtful case the patient should be anesthetized and a ureteral catheter passed to ascertain whether there is an obstruction and whether relief can be given. The diagnosis can thus be cleared.

It is self-evident that recurring dilatations of the pelvis of the kidney or more or less permanent hydronephrosis should not be allowed to occur as the kidney structure may become permanently diseased, pyelitis may result, and the kidney may become useless. It should also be remembered that serious irritation or inflammation of one kidney cannot occur without danger of involving the other kidney.

Of course it is recognized that when the abdomen is fat, ordinary physical examinations and even ordinary roentgenograms of the abdomen may not make the diagnosis clear, and that cystoscopy, ureteral catheterization, pyelography, and perhaps even exploratory laparotomy are necessary to make the diagnosis positive.

URETERAL CATHETERIZATION AND PYELOGRAPHY

CATHETERIZATION OF THE URETERS

If pus or blood in the urine is not positively known to come from the urethra, the source of either one or both must be ascertained, and the diagnosis of the cause will determine the treatment.

As it is not proposed in this discussion of kidney disturbances to describe diseased conditions of the urethra, bladder or the genital adnexa, urethral, bladder, prostate and seminal vesicle tests will be only briefly mentioned. The two and three glass office tests in gonorrhea should be routine. Young's seven glass test, well described by Thomas,[120] should be done from time to time in male cases to determine the source of the pus and what parts of the genito-urinary system are involved. If the case is not one of gonorrhea, then these tests should be carried out immediately as a prelude to more accurate physical examinations. It may also be stated that an organism, apparently a gonococcus under the microscope, does not positively prove that gonorrhea is present unless gonococci can be shown in cultures and unless the so-called complement-fixation test is positive. It should also be noted that the complement-fixation test does not appear until the posterior urethra is infected in the male and the cervix in the female, and then not until the disease has been present four or more weeks. If the test is negative, it does not prove that gonorrhea is not present. The fixation test may be positive weeks after an apparent cure, proving that gonococci probably are present somewhere in the organism.

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[120] Thomas: Am. Jour. Med. Sc., November, 1913, p. 696.

Whether gonococci have migrated to a joint, a kidney, or to some other region, a gonococcic vaccine injection may give a focal reaction of value in diagnosis. As a cure it is not a great success in acute gonorrhea or in its complications, but it may be of assistance in secondary subacute cases. An autogenous vaccine may be of supplemental value to other treatments.

Microscopic examination of secretion from a massaged prostate, culture examinations of sediment of the excretion (pus, blood, mucus and semen) obtained by an injection into a guinea-pig if tuberculosis is suspected, urethroscopy, cystoscopy, the passage of catheters into each ureter if necessary, roentgenography, pyelography, the tuberculin diagnostic test, and the Wassermann reaction should reveal the origin of pus or blood, or both, in the urine.

It is now possible to shorten the period (about six weeks) of the guinea-pig test of secretion or excretion suspected of being tuberculous. Murphy and Ellis[121] have shown that when white mice are exposed to Roentgen rays they are more susceptible to bovine tuberculosis than the normal animal, and Morton[122] of the Massachusetts General Hospital has shown that the resistance of guinea-pigs to tuberculosis is reduced by Roentgen-ray exposures. Therefore the diagnosis of suspected renal tuberculosis can be ascertained in from eight to ten days by the inoculation of a guinea-pig exposed to Roentgen rays. The application of the Roentgen ray to the guinea-pig for this object may be made in one large dose administered just before or shortly after the animal is inoculated.

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121. Murphy and Ellis: Jour. Exper. Med., 1914, **20**, 397.
122. Morton: Jour. Exper. Med., 1916, **24**, 419.

The ureters rarely show primary disease, but acquire disease or disability from above (the kidneys), less frequently from below (the bladder), or occasionally from some disease or condition external to them.

It is hardly necessary to express the caution that before the passing of ureteral catheters the bladder should be thoroughly washed, lest infection be carried into the healthy ureter and to a healthy kidney. When the bladder is seriously infected, a ureteral catheter should perhaps not be passed at all unless urgently necessary.

As stated in the discussion on bleeding from the kidney, the term "essential hematuria" should not be applied unless all other causes have been eliminated, and even then surgery may find a tangible pathologic excuse for such bleeding. A previous lesion, followed by a cicatrix, and the formation of varicosities and consequent bleeding are tangible causes of so-called essential hematuria, and cases have recently been described by Payne and MacNider,[123] showing that some such cause may be at the base of many if not of all so-called essential hematurias. Of course there may be hemophilic bleeding from any portion of the genito-urinary tract without previous local disease.

PYELOGRAPHY

While in many cases the usefulness of this method of obtaining roentgenograms cannot be disputed, it should be emphasized that only by the exercise of great care is the method free from danger. It seems essential for safety that the opaque substance, colloidal silver or whatever it may be, should run into the pelvis of the kidney by gravity only and not by any syringe pressure. If pain occurs, the instilled sub-

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123. Payne, R. L., and MacNider, W. B.: The Surgical Problem of Unilateral Symptomless Hematuria, THE JOURNAL A. M. A., Sept. 23, 1916, p. 918.

stance has apparently caused sufficient distention of the pelvis to force the solution into the terminal kidney tubules.

Therefore this ultimatum of scientific roentgenography should not be resorted to unless other simple means of diagnosis have failed. If the renal excretion is shown by various tests to be decidedly insufficient, and if the patient is in a weakened physical condition, pyelography should not be attempted.

The objects of pyelography are: to locate a kidney as to its relation to some tumor; to determine positively the amount of mobility of a kidney (roentgenograms being taken with the patient standing and lying); to accentuate a doubtful roentgenogram of a renal calculus; and to determine accurately the dilatation of a kidney pelvis in hydronephrosis and in pyonephrosis.

While the normal capacity of the pelvis of a kidney is not entirely agreed on by surgeons who specialize in diseases of the kidney, testimony seems to show that any capacity below 30 c.c. (an ounce) may be normal; any capacity above this amount is generally abnormal, and some patients complain of pain after the instillation of 15 c.c. (half an ounce) of liquid. Overdistention of a kidney, whatever the size of the pelvis, and rapid distention will cause pain and may cause shock. A rise of temperature is not infrequent after pyelography. Childs and Spitzer[124] state that it should also be recognized that considerable twisting of the ureter may not be abnormal.

An efficient technic of pyelography can be acquired only by association with some expert in this line of work. It is obvious that the technic can be carried out only in a hospital with proper Roentgen ray equipment. The patient who is to undergo the examination should be physically and renally able to endure it. He should rest for two days before the operation — for it is an operation. During these two days his diet should be rather light, and sufficient purging should be caused to obtain the best picture results. Several opaque silver preparations are used in pyelography. It is not clear that any one is preeminently the best. Collargol has been, perhaps, the most frequently used, but thorium nitrate in 20 per cent. solution has more recently been advocated.[125] Whatever solution is used, it should be entirely evacuated in twenty-four hours. In some cases the silver solution has been found retained in some part of the kidney structure.

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[124] Childs, S. B., and Spitzer, W. M.: Roentgenographic Study of the Normal Kidney, Its Pelvis and Ureter, THE JOURNAL A. M. A., Sept. 20, 1913, p. 925.

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[125] Burns, J. E.: Thorium—A New Agent for Pyelography, The Journal A. M. A., June 26, 1915, p. 2126; Further Observations on the Use of Thorium in Pyelography, ibid., Feb. 17, 1917, p. 533. Braasch and Mann: Am. Jour. Med. Sc., September, 1916, p. 336.

PYELITIS

Pyelitis develops from any obstruction of a ureter, whether of external or internal cause; from calculi, blood or bacteria in the pelvis of the kidney; from infection of the kidney substance; from tuberculosis of a kidney; from adjacent suppuration, and from an ascending infection of the bladder or pelvic organs.

If the ureter is not obstructed, germs reaching the kidney and being rapidly excreted, or even the reproduction of certain germs in the pelvis of the kidney, such as colon bacilli or typhoid bacilli, may not cause pyelitis. If, however, the ureter is obstructed, if the canal is imperfect, or if there is a chronic irritation or inflammation of the pelvis of the kidney, pyelitis sooner or later occurs. Infection or pyelitis of one kidney is a menace to the other.

Intestinal upsets, especially when aggravated by frequent constipation, allows the colon bacillus and other germs to reach the kidney through the lymphatics, and not infrequently in these conditions colon bacilli are found in the urine. These may disappear on the correction of the intestinal stasis. At times the colon bacilli may reach the kidney by way of the blood.

Direct ascending infection from the bladder through the ureter to the kidney is not frequent, except when there is urethra or bladder obstruction, such as an enlarged prostate. Infection of the bladder and pelvic organs may reach the kidneys through the lymphatics. This has recently been shown by Eisendrath and Schultz.[126]

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[126] Eisendrath, D. N., and Schultz, O. T.: Lymphogenous Infection of the Urinary Tract, The Journal A. M. A., Feb. 17, 1917, p. 540.

While in the majority of cases the colon bacillus is the germ of infection, the tubercle bacillus, the typhoid bacillus and the various pus-producing germs are also causes of pyelitis, and, of course, frequently more than one variety of germ is present.

Females have infected kidneys more frequently than males; this is especially true in young children. The exact reason is not clear. Though the ease with which vaginal infection reaches the bladder may be a factor in the cause of kidney infection, in pregnant women it is doubtless due to some ureteral obstruction from pressure or twisting with the consequent dilatation of the renal pelvis; as stated, such a kidney is susceptible to infection.

SYMPTOMS

A kidney infection may cause only a bacilluria as evidenced by a slightly cloudy urine with a peculiar fishy odor; or it may cause a slight cystitis, with cloudy urine and an increased amount of mucus, perhaps followed by pus. Only cystoscopy and the passage of the ureteral catheter will determine whether one or both kidneys are affected. In these cases, if the urine is strongly acid there is less bacilluria and the urine is less cloudy than when it is alkaline in reaction. Localized pain, a loose kidney, or a palpable enlarged kidney may presumably show which kidney is affected. It is probably rarely found that pus comes from the two kidneys at once, but a purulent infection of one kidney sooner or later causes inflammatory or other disturbances of the other kidney.

A colon infection and bacilluria may continue for months without the development of pus and without causing many symptoms, but if pus is issuing from a kidney, there is almost invariably a history of pain, with colicky ureteral pains, in an exacerbation of which blocking of the ureter often occurs. The urine at this time coming from the healthy kidney may be clear. After the urine and pus back of a blocked ureter force a passage, a large amount of pus suddenly appears in the urine. While the pus is being retained and not freely evacuated from the kidney, fever generally occurs, often preceded by chills, rigors, and frequently by attacks of profuse sweating. Repetition of this history is typical of a purulent pyelitis.

These purulent urines from a kidney source are generally acid in reaction, and contain albumin often more than can be accounted for by the pus, and, sooner or later, casts. Frequently, however, there may be no albumin in the urine after the pus has been filtered out.

There is usually progressive loss of weight, loss of appetite, indigestion, restlessness during the night, and finally anemia. A somewhat constantly recurring afternoon or evening rise of temperature suggests a tuberculous infection, while an ordinary septic infection generally causes fever only when there is an obstruction to the free flow of pus. Hence a purulent pyelitis may go on for months without apparent fever.

It has long been recognized that typhoid bacilli may be found in the urine for weeks or even months after typhoid fever. It has been shown also that not infrequently tubercle bacilli may appear in the urine when there is a tuberculosis of some part of the body, especially of the lungs, even though the kidneys may not be involved. These bacteria, unless associated with secondary infection, may not produce symptoms, and do not cause pus. Doubtless colon bacilli occur in the urine on account of some stasis or intestinal indigestion much more frequently than is now known, and probably generally without causing symptoms.

The amount of pus in the urine may diminish and entirely disappear for a time and then recur; in the interim, however, the urine, if cultivated, may show germs.

TREATMENT OF PYELITIS

The diagnosis of these infections of the kidney can be determined only by the microscope, by staining the bacteria, and by cultures; which kidney, if not both, is involved must be determined by the physical examination, by the passage of the ureteral catheter, by roentgenograms and, if necessary, by pyelography. All primary infections or localized infections in other parts of the body must be discovered and removed, if possible.

Whatever the germ or germs may be that are causing the infection, the objects aimed at are: (1) good circulation in the kidneys; (2) prevention of absorption of toxins and germs from the intestine; (3) free drainage, and (4) modification of the character of the urine.

1. It is essential that the circulation in the involved kidney should be as perfect as possible; hence, if the kidney is displaced, it should be held in proper position. If the general circulation is poor, it should be improved by digitalis or by some other method. Ordinarily, it is not advisable for such a patient to remain in bed, as bed rest tends to increase the congestion of the kidneys; better drainage from the pelvis of the kidney is secured when the patient is up and about.

2. The bowels should be moved freely every day. Whether a saline laxative or a vegetable laxative should be used will be determined by the needs of the individual patient. The diet should be one which tends to cause the least intestinal fermentation. It may be

advisable temporarily to change the diet radically from a mixed to a vegetable, or a milk diet. It may be well to give yeast or lactic acid bacilli.

3. To meet the third indication the patient should drink plenty of water to dilute the urine and to wash out more effectively any débris that may have been deposited in the pelvis of the kidney, whether bacteria alone, or pus, mucus and bacteria.

4. If the urine is very acid, especially if there is bladder irritability, it may be well to give an alkali for a short time, such as potassium citrate in 2 gm. doses; but this alkali should not be given over a long period of time, nor should the urine be allowed to be long alkaline, as most germs grow better in an alkaline medium. Sometimes much of the bacilluria disappears when the urine is made normally acid. Hence if the urine is alkaline in a bacilluria (which is generally not true when there is kidney pus), dilute hydrochloric acid should be given after meals. The dose need not be large — from 5 to 10 drops.

Perhaps the best germicidal treatment is hexamethylenamin, in a dose of 0.5 gm. (7½ grains) in water four times a day. It has been stated that to be antiseptic in the pelvis of the kidney the dose of hexamethylenamin must be much larger than this, but most persons tolerate a larger dose than the one named only for a short time. This drug may cause some bleeding from the kidneys, uretal pain and bladder irritation. When hexamethylenamin is prescribed, an alkali should not be given.

If this treatment, which is the best, does not succeed, or if for any reason hexamethylenamin disagrees with the patient, phenyl salicylate may be given in 0.25 gm. doses, in capsule form, four times a day. This is sometimes successful in mild cases of bacilluria. Santal oil has also been used.

This treatment is frequently successful in more or less permanently curing a bacilluria; at times it is used with success in treating a mild purulent infection of the pelvis; and it is also possible to evacuate and heal a small abscess of a kidney in this manner.

On the other hand, most purulent kidneys are difficult to treat. Both stock and autogenous vaccines have been used with varying success; perhaps the most usual result is failure.

A more radical treatment is to wash out the pelvis of the kidney through the ureteral catheter with boric acid or a silver solution.

Geraghty[127] finds that in simple pyelitis renal pelvis lavage is of great value, and he uses either a silver nitrate solution or a weak solution of formaldehyd. He injects into the kidney pelvis from 5 to 10 c.c. of a 0.5 per cent. silver nitrate solution, and passes the catheter only a short distance into the ureter for the purpose of treating a coincident ureteritis at the same time. The strength of the injection is gradually increased until it has reached 5 per cent. The increase is gradual to obtain tolerance.

It can be readily understood how frequently little pockets of pus — which may be discovered by pyelography — may be located in the kidney tissue around the pelvis. When such small abscesses are present, even with injections, the cure may be slow and imperfect, and they may readily recur.

Geraghty believes that when there is considerable pelvic dilatation, pelvic lavage is of little value. He has also advantageously used formaldehyd solution, from 1 part to 5,000 parts of water, to 1 part to 2,000 parts of water.

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[127] Geraghty, J. T.: The Treatment of Chronic Pyelitis, The Journal A. M. A., Dec. 19, 1914, p. 2211.

Kretschmer and Gaarde[128] believe that many cases of pyelitis in adults, especially when it occurs during pregnancy, represent exacerbations of a pyelitis of infancy which has remained latent for years. They believe that chronic colon pyelitis which has resisted medicinal treatment is best treated by pelvic lavage with a 1 per cent. solution of silver nitrate; they reject the stronger solutions suggested by Geraghty. If there is much pus in the pelvis of the kidney they wash it out first with a weak boric acid solution. They use, on an average, from 5 to 7 c.c. of a 1 per cent. silver nitrate solution, and repeat the injections at five or six day intervals until the urine is sterile and free from pus. The number of injections required in each case in the fourteen cases of pelvic lavage summarized varied from one to eight. In these fourteen cases they reported eleven cures, as proved by negative cultures. All injections must be given slowly, and actual distention of the pelvis avoided.

Murphy[129] believed that though vaccines often fail, they are frequently of great value in genito-urinary infections. He condemned their indiscriminate use, however, as in obstructive pyelitis, but advised the use of vaccines in infection with free drainage when the condition of the patient was satisfactory. He recommended caution in dosage, and began with 100 million dead bacteria as the first dose, increasing this number rapidly until there was a reaction. He stopped the treatment from three to eight days and then gave the same or a slightly increased dose, according to the previous reaction. A severe headache or a temperature of 102 indicates a smaller dose, and the largest dose should not exceed 30 billion bacteria.

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128. Kretschmer, H. L., and Gaarde, F. W.: The Treatment of Chronic Colon Bacillus Pyelitis by Pelvic Lavage, The Journal A. M. A., June 24, 1916, p. 2052.

129. Murphy, J. B.: Interstate Med. Jour., November, 1914, p. 1214.

The injections should be given deep into the muscles, and slowly. When more than 10 billions are given at once they should be injected at two or three separate points. Of course smaller doses may be given intravenously. A severe local reaction should not alarm, although these injections may cause necrosis of tissue.

Murphy obtained the best results in colon bacillus infections. He believed that if the infection of the kidney came by way of the blood, the trouble would be in the parenchyma of the kidney and the microscopic examination of the urine might suggest the depth of the location of the infection in the kidney substance by the character of the epithelial cells. If an infection reached the kidney from below by way of a dammed back urine, the pelvis would of course be infected first. He also recognized the lymphatic route of infection from the pelvis or other parts of the abdomen.

If an autogenous vaccine cannot be produced from the infecting bacteria in the urine or from some localized infection, like that of the prostate, it may be isolated from the blood serum or from an infection in some other part of the body, if such causative localized infection is present. The tonsils, the sinuses adjacent to the nose, and the gums and teeth should be studied for possible sources of infection.

While the most frequent germ of infection is the colon bacillus, the staphylococcus, the streptococcus and the gonococcus are also not infrequent infective agents. Ramsey[130] found that in 100 cases of infection, ninety were caused by the colon bacillus.

If a purulent kidney does not improve rapidly, if the patient is becoming debilitated, or if the kidney is found enlarged and examination of the urine from this kidney shows that the kidney structure is diseased, temporizing should cease, and the kidney should be removed, unless the other kidney is so diseased as to render the operative danger very great.

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[130] Ramsey: St. Paul Med. Jour., 1914, **16**, 343.

POSTOPERATIVE RENAL INFECTION

This should be suspected when, during convalescence, the patient has a sudden chill, mild or severe, a rise of temperature, with or without pain in the affected kidney, and head symptoms, followed by irregular and remittent fever. This is true, of course, only when more obvious causes of these symptoms are excluded. The urine is at first scanty; later it may show albumin, blood, pus, casts and colon bacilli or other bacteria.

PYELITIS IN INFANCY

This disease is about three times as frequent in girl as in boy babies, and the colon bacillus is by far the most frequent germ of infection found. Smith[131] states that the pelvis of the kidney is the part most commonly affected, with the ureters and bladder generally normal. Some cases may show inflammatory changes in the kidney substance, due to extension from the pelvis of the kidney. Even in children, the right kidney seems to be the one most frequently affected in unilateral infection.

Smith gives a long list of references, but thus sums up the mode of infection:

> From the intestinal tract or some other source bacteria get into the lymphatic vessels and then into the blood, or possibly directly into the blood. They are transferred by the blood to the kidney. After reaching the kidney they pass through the glomeruli and are excreted at the pelvis. They may pass out of the body in the urine without doing any damage, or they may set up an infection at the point of excretion, that is, produce a pyelitis.

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[131] Smith, R. M.: Pyelitis of Infancy, Am. Jour. Dis. Child., September 1916, p. 235.

The colon bacillus probably comes directly from the intestinal tract. Smith and other writers agree that the gastro-intestinal tract should be properly treated in pyelitis, not only when the colon bacillus is the source of infection, for, as in adults, other bacteria may reach the kidneys and cause infection from such sources as the tonsils. Not only typhoid bacilli and tubercle bacilli, but also colon bacilli may pass through the kidney substance without causing any localized infection. While it is not thought that colon bacilli or other bacteria often travel upward, even in women, some injury may cause absorption of these germs, which are so constantly present in the genital organs of females.

There is a difference of opinion as to the frequency with which colon bacilli occur in the urethra of female children and of women. Alsberg[132] found colon bacilli in the urethra of adult women in 100 per cent. of the cases. On the other hand, Beeler and Helmholz[133] say that while pyelitis is much more common in young girls than in boys, the colon bacillus is not normal in the female urethra, and they came to this belief after examining thirty infant girls and thirty-one girls over 2 years of age. In infections not genito-urinary, in girls under 2 years of age, the colon bacilli are frequently found in the urethra.

The symptoms of a pyelitis in a child are well described by Smith,[134] who states that the infection begins in a child with a chill and fever which is relapsing or remittent. He finds that a chill occurring in an infant under 2 years of age is very suggestive of this disease.

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[132] Alsberg: Arch. f. Gynäk., 1910, **90,** 255.

[133] Beeler, Carol, and Helmholz, H. F.; The Bacteriology of the Urine in Healthy Children and Those Suffering from Extra-Urinary Infections, Am. Jour. Dis. Child., October, 1916, p. 345.

[134] Smith, R. M.: Recent Contributions to the Study of Pyelitis in Infancy, Am. Jour. Dis. Child, March, 1913, p. 273.

The nervous phenomena occurring in the beginning of this infection may even suggest a meningitis. There is apt to be frequent and painful micturition, but perhaps no other genito-urinary signs. Sometimes the patient is very pale and the abdomen tender, or a kidney region may be tender, while often the spleen is enlarged. There may be joint tenderness and herpes. With recurrences of fever there may be vomiting, and even coma may occur. The urine may show pus, or it may be clear if the ureter is blocked by the pus; hence every urinary excretion must be caught and examined in order to make the diagnosis, as the purulent urine is apt to be intermittent. There may be albumin in the urine.

Smith says that the prognosis is generally good, that the disease is rarely fatal. The acute symptoms, however, may last several weeks, and the recovery may be protracted; or the infection may persist, or recurrences may take place for years. Later the bladder may become infected, and there may be abscesses in the kidney structure. There may be a history of sore throat or some intestinal upset occurring immediately before this infection.

The treatment consists in locating, and removing if possible, the causative infection. Purgatives should be given, and colon washings are often advisable. A liberal amount of water should be given the patient, so as to encourage a free outflow of pus. Alkalies, such as potassium citrate, are advisable, given in the dose and frequency sufficient to alkalize the urine. As cautioned with adults, the urine should not be made alkaline for too long a time; or, if it is found alkaline (which is rare), it should be made acid. Hexamethylenamin in proper dosage for the age of the patient may be tried, as it will sometimes be found of advantage, although it often fails, especially in colon infection. Vaccines may be tried if deemed advisable and if the disease is protracted; surgical measures may be necessary.

HEMATOGENOUS INFECTION OF THE KIDNEYS

In 1906, Brewer[135] first described this condition as a distinct pathologic entity. In a series of subsequent communications he has clarified the understanding of this not infrequent condition, and shown the best treatment for it. One of his latest papers[136] brings the subject nearly up to date.

Pathologically, the local lesion is caused by the plugging of one or more blood vessels, with groups of organisms acting as emboli. Inflammatory and protective processes begin around this infarct, and the center becomes a minute abscess. Several of these plugs and subsequent abscesses may run together and form larger abscesses, or a plug of a larger arteriole may cause a large section of the kidney to break down at once. If the condition is neglected, these larger abscesses may not break into the urinary tubules or into the kidney pelvis and be thus evacuated, but may rupture the kidney capsule and form a perinephritic abscess.

The origin of these localized kidney infections may be a serious infective process or some simple infection, such as a boil, a carbuncle, tonsillitis, a tooth or sinus infection, or an infected wound. The infarcts in the kidneys may be bilateral, but are frequently unilateral.

Brewer recognizes three types of hemic kidney infection: The first is the serious and fulminating type, which is rare. In this form there are symptoms of acute general infection, with the kidney signs very hazy, and death may occur almost without a kidney symptom. However, when both kidneys are affected there may be total suppression of urine, rapid prostration, and death will result from sepsis and uremia. When only one kidney is thus seriously attacked, death is due to sepsis.

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[135] Brewer: Surgery, Gynec. and Obst., May, 1906, p. 485.
[136] Brewer: New York Med. Jour., March 20, 1915, p. 556.

The second type is serious, but does not become acute as rapidly; however, the prognosis is bad, although the disease reaches a fatal issue more slowly. In this type the progress is so rapid and the local signs may be so few that the whole kidney may be destroyed before a diagnosis has been made, while the function of the other kidney also may become impaired.

The third is a milder type in which the condition occurs and the patient recovers often without a diagnosis. This type of mild infection frequently accounts for slight unexplained rises of temperature, followed after some weeks by an unaccountable pyelitis.

This disease or kidney infection may not begin with a chill, unless it is of the severer type, but the temperature may rise to 103 or 105, with a pulse rate of 120. The toxemia is severe, and the general condition of the patient may resemble a severe hidden or central pneumonia. Soon he complains of vague pains in the abdomen or in the flank on the affected side. If the right kidney is affected, the symptoms and signs may resemble an acute appendicitis or an acute gallbladder infection, and from these conditions it may be difficult to make a differential diagnosis. The affected kidney may secrete but little urine, while the other kidney may secrete freely, or even more urine than normal. Careful examination of the urine, however, may show traces of albumin, blood and pus, and in doubtful conditions the ureters should be catheterized. Brewer considers the presence of a decided unilateral costovertebral tenderness a pathogenic sign. In these acute cases he believes the only safe treatment is immediate nephrectomy.

The milder forms of this infection, which are more frequent, often resemble appendicitis, cholecystitis, or abscess of the liver.

If the disease is not recognized and not surgically treated, it progresses to the formation of large abscesses, as just stated, and even to pyonephrosis. These abscesses are smaller and fewer than occur in the acute or fulminating type, and cutting down on the kidney, stripping the capsule, and opening and draining the cortical abscesses may often save the kidney. A neglected case may require complete nephrectomy. When a diagnosis has been made the treatment is rest, plenty of water, and hexamethylenamin.

Brewer believes the reason this disease is so generally unilateral is that a well kidney may throw off such an infection, while a kidney which has had some previous lesion or disturbance, or a kidney that is loose or mobile, is more susceptible to infection. He has isolated the *Bacillus coli,* the *Streptococcus pyogenes,* the *Staphylococcus pyogenes-aureus* and the *Bacillus typhosus* in these cases. He has come to the conclusion that hematogenous infection is doubtless the cause of the greater number of renal sepsis cases.

TUBERCULOSIS OF THE KIDNEY

The kidney is not an infrequent location of this disease; unfortunately, however, often the disease is not discovered until a septic infection has been added to the primary condition, that is, pyelitis has occurred. It occurs most frequently at the age when general tuberculosis occurs, namely, from adolescence to 35 or 40 years of age. In the beginning the disease is probably always unilateral, and ordinarily the other kidney seldom becomes involved, unless the patient has been seriously neglected. This disease of the kidney is often secondary but frequently it is a primary infection; hence the urgency of early diagnosis and its eradication lest the disease generalize and affect other parts of the body.

The pathology is that of tuberculous lesions — first small points of infection, then nodules, later destruction, and finally abscess formation. The pathologic findings seem to show that while certain lesions may be evacuated and become cured, or may heal as do tuberculous deposits in the lungs, still other parts of the kidney show continued tuberculous degeneration. In other words, there is no proof that a tuberculous kidney ever becomes permanently or safely healed; consequently, the only treatment, after the diagnosis has once been made, is nephrectomy.

Probably the involvement of the ureter or of the bladder is always secondary, and such infection would not have occurred had the diagnosis been made early.

The usual symptoms are: pain, not always a factor, as there may be only a sense of discomfort in the abdomen, more noticeable on the affected side; backache; a frequent desire to urinate, and as the disease progresses, usually a slight amount of blood in the urine, or a real hematuria. Soon pus appears.

As these symptoms progress, there is generally afternoon or evening fever, possibly night sweats, loss of appetite, loss of weight, and general debility. Varying amounts of albumin and casts are usually found in the urine. There may be occasional attacks of colic caused by obstruction of the ureter with pus or débris, with the usual symptoms of rise in temperature, pain, possibly vomiting and chills, and later evacuation of urine that is loaded with pus.

Blood or pus in the urine, as repeatedly stated in this discussion, necessitates examinations to determine the source. The frequency of tuberculosis of the kidney should cause one to suspect this disease when pus or blood, or both, appear in the urine. Physical examination may readily show which kidney is affected. Carefully cleansing the genitals of all smegma, centrifuging the sediment of the suspected urine, and staining for tubercle bacilli will frequently reveal the germ, if it is present. Or, the urine obtained by a catheter passed into the ureter of the involved kidney may be thus treated. Failure to find tubercle bacilli by these methods does not prove that the disease is not tuberculosis. The guinea-pig test with the sedimented, centrifuged urine is apparently reliable, and the number of weeks necessary for such a test to give a positive or negative result may be shortened by exposing the injected animal to the Roentgen ray, as has been shown by Murphy and Ellis[137] and by Morton.[138] Observation of the temperature of the patient for several days will aid in deciding whether or not the kidney is tuberculous.

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[137] Murphy and Ellis: Jour. Exper. Med., 1914, **20**, 397.

[138] Morton: Jour. Exper. Med., 1916, **24**, 419.

If the kidney is tuberculous, there is no effective treatment except nephrectomy, and the ureter, if infected, should be removed as low down as possible. Tubercle bacilli may continue to appear in the urine from an infected ureter or infected bladder for weeks or even months after the operation, but they generally disappear. Of course if the other kidney is diseased, the operation would not be advisable, but, as stated, the two kidneys are rarely affected with tuberculosis at the same time. After the operation the remaining kidney slowly hypertrophies, and within several months is able to carry on the ordinary functions of both kidneys.

When the offending organ has been removed the patient should for a long time be treated as though affected with pulmonary or general tuberculosis. This means rest, fresh air, good food, the prevention of such exertion as will cause fever or loss of weight, and such exercise or overfeeding as will overtax the kidney before it is able to do its double work. He also generally requires iron for some time. Vesical irritability may be relieved by alkalies, if the urine is acid. If there is considerable infection of the bladder, it may be carefully washed out for a time. Phenyl salicylate may be given in small doses; hexamethylenamin, if used, should be given in small doses, as great care is necessary in administering any drug which tends to irritate the remaining kidney.

TUMORS AND CYSTS

MALIGNANT TUMOR OF THE KIDNEY

The cardinal symptoms of cancer and sarcoma of the kidney are pain, blood in the urine, and palpable enlargement of the kidney. The pain may be local, or may be referred to different parts of the abdomen, to the back, to the epigastrium, or downward. If the right kidney is involved, the disturbance must be dissociated from a gallbladder infection. The pain may be dull, boring, or burning in character. In occasional cases there is no pain except that of the dragging of the tumor, which may be alleviated by a support of the abdomen and the tumor. Sometimes there are shooting nerve pains like an intercostal abdominal neuralgia or neuritis.

In the majority of cases, blood is found in the urine, and by the time the patient seeks help, there is generally a tumor mass. A tumor in the kidney region may be of some other organ than the kidney, it may have displaced a kidney, or it may be an abnormally large kidney from hydronephrosis or pyonephrosis. The centrifuged urine will generally show not only blood, but also pus and cells of broken down tissue, often characteristic of the growth. With a demonstrable tumor and the urine showing this pathologic condition, the affected kidney is clearly revealed. Of course, catheterization of the ureters not only will give more concentrated pathologic findings in the urine, but also will show the condition of the other kidney.

The general condition of the patient is an important indication of the gravity of the lesion. There may be secondary disturbances, weakness, anemia, emaciation, and gastro-intestinal abnormalities.

Braasch[139] finds that renal growths are more frequent in men than in women.

Renal function tests and blood tests should always be made and the efficiency of the other kidney determined before a nephrectomy is done; pyelography is occasionally necessary.

Whether or not life will be prolonged by nephrectomy depends on how far the case has progressed. As so often repeated, every bleeding into the urine should be investigated in order that an early diagnosis of a kidney growth may be made before an operation is of no avail. While cancer of the kidney may be primary, in the majority of cases it is secondary.

Carcinoma of the kidney causes a great deal more pain than does tuberculosis, and the progress of the disease is much more rapid, although, as stated above, it is sometimes devoid of pain. Colicky pains may occur when small clumps of degenerated tissue or blood clots pass down the ureter.

Sarcoma of the kidney is perhaps more frequent than carcinoma, and the progress to a fatal issue more rapid. Like carcinoma or sarcoma in any other part of the body, and perhaps particularly when affecting the kidney, on account of late discovery, recurrence of these growths after nephrectomy is frequent.

HYPERNEPHROMA

This growth of the kidney is more frequent than has been recognized. It has a tendency to cause metastases and although often accompanied by pain, profuse bleeding may be the only symptom. Generally there is a palpable tumor. When cystoscopy reveals hemorrhage and enlargement of one kidney, when there is no pus in the urine, and no symptomatic or roentgenographic evidence of stone, hypernephroma or some other malignant growth should be diagnosed, and an early operation is necessary to avoid metastases and recurrence of the growth.

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[139] Braasch, W. F.: Clinical Data on Malignant Renal Tumors, The Journal A. M. A., Jan. 25, 1913, p. 274.

CYSTIC DISEASE OF THE KIDNEY

In this condition there are multiple cysts in the kidney structure, and one or both kidneys may be involved. The symptoms are enlargement, recurrent pains, and more or less profuse hematuria. Sometimes these patients live for many years and endure well the loss of blood; others quickly become anemic; still others develop serious insufficiency of the kidneys. Surgical cure, even when only one kidney is involved, cannot be promised, as the mortality is great, and when both kidneys are involved, surgical interference should probably not be attempted.

Congenital small cysts may occur on the kidneys, and perhaps cause no symptoms for a long time. However, these nodular kidneys increase in size and become tumor masses, and sooner or later the kidney structure becomes involved, resulting in kidney insufficiency.

THE KIDNEYS IN PREGNANCY

It has long been recognized that in pregnancy the function of the kidneys is easily disturbed and that their ability to excrete properly during this period of increased metabolism is frequently more or less impaired. The circulation of the kidneys is readily disturbed by the enlarging uterine tumor. A previously loosened kidney may have its ureter twisted, and interference with the circulation of the kidney may cause passive congestion, and the appearance of albumin in the urine.

Wilson[140] has recently found that normally pregnant women begin to store nitrogen early in pregnancy, though this reserve may be used up in the puerperium and during lactation, and Werner,[141] after extensive tests, found that the functional capacity of the kidneys of healthy pregnant and parturient women is somewhat impaired. Hence any interference with kidney excretion is more dangerous in a pregnant woman than in other persons. Consequently, temporizing with an albuminuria, with or without casts, and with retained nonprotein nitrogen during pregnancy is apparently inexcusable, unless the patient is near the termination of her pregnancy, and she is under careful observation.

The thyroid gland normally hypersecretes during pregnancy. If for any reason it does not perform this office, and undersecretes, or does not secrete sufficiently for the double metabolism of mother and child, nitrogen or possibly ammonia poisoning readily

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140. Wilson: Bull. Johns Hopkins Hosp., May, 1916.
141. Werner: Arch. f. Gynäkol., 1915, **104,** No. 3.

occurs, and such patients may become eclamptic, although albumin or casts may not appear in the urine. When the ammonia nitrogen of the urine, which is normally about 5 per cent., reaches 16 per cent. or more, as in pernicious vomiting and consequent starvation, the uterus should be emptied unless starchy food in some form can be given and retained.

A woman who has shown a previous insufficiency of the thyroid or disturbance of the kidneys, or who has given birth to children who have shown subthyroid conditions, should receive thyroid from the beginning of a new pregnancy, the dose depending on its effect and her need for such treatment. Frequently with this careful thyroid medication such a woman will go through pregnancy normally and give birth to a normal child.

NEPHRITIS IN PREGNANCY

Of course all forms of nephritis may occur in pregnancy, owing to any of the usual causes; hence an acute nephritis or a subacute nephritis may occur, and cannot be distinguished from the same condition when due to pregnancy; or pregnancy may revive a supposedly cured nephritis. There is also no doubt that a woman who has had nephritis during pregnancy is more liable to have a recurrence in subsequent pregnancies. Some kidneys, possibly because of their position, mobility, or the position of their blood supply, are readily impaired by the abdominal pressure of the enlarging uterus. A chronic nephritis is intensified with pregnancy.

The occurrence of albuminuric retinitis in a pregnant woman is very serious, and the disturbance may be permanent, even if the uterus is immediately emptied, although the patient may completely recover.

Even if a patient with nephritis is apparently doing well and is allowed to go to normal time for labor, the exertion of labor, which throws more nitrogen, especially creatin and acids, into the circulation may cause rapid development of convulsions and coma. She is on the borderline of uremic poisoning, and the extra muscular exertion is the last straw needed to cause the uremic attack.

Nephritis may cause abortion, and the child is often born dead; but if a child is born of a seriously nephritic patient, it is liable to be anemic and of low vitality.

To summarize: albumin and casts do not occur normally in pregnant women, although they may occur accidentally, as in any other person. If they persist, the prognosis is bad. Functional tests may be made, and the nonprotein nitrogen of the blood noted; but the future is doubtful, even when all these tests are normal. At parturition, labor will often be sluggish, especially if there is edema of the parts involved. The uterus does not well contract, and postpartum hemorrhage is frequent.

While it is generally advisable to give an anesthetic so that the patient will not use her muscles so strongly and not force as much urea and creatin into her blood, the anesthetic is an added irritant to the kidneys. The period of anesthesia should, therefore, be as short as possible, and the uterus should be emptied as rapidly as possible.

If nephritis occurs early in the pregnancy there is no question of the necessity for immediate cleaning out of the uterus; postponement is dangerous. If nephritis occurs in the latter stages of pregnancy, if there is no retinitis and no anasarca, and if the circulation is good, with rest, proper diet and constant study of the nonprotein nitrogen of the blood, "watchful waiting" is permissible, but it is always a question necessitating careful decision, and consultation should be sought, to divide the responsibility. A woman with chronic nephritis should not marry, or be allowed to become pregnant if nephritis should occur after marriage.

If the symptoms do not improve after the uterus is emptied decapsulation should be considered, and should be seriously considered during eclampsia.

DISTURBANCES OF THE BLADDER

URINARY STASIS

This condition occurs more frequently than has been recognized, and when urine is retained, bacilluria readily follows. While normally the female bladder empties itself completely, it does not do so in many pelvic conditions. This is especially true in pregnancy, which probably accounts for the frequency of genito-urinary infection during and after that condition. Colon bacilli seem frequently to be found in the urine in pregnancy. A pyelitis may occur during pregnancy from an ascending bladder infection or from lymphatic absorption of any pelvic infection. This is one more reason for a frequent examination of the urine during pregnancy. If the kidney functions are found normal, a bacilluria should immediately suggest a bladder insufficiency.

Vesical irritation and urinary symptoms may be caused by rectal disturbances, as shown by Zobel.[142]

INCONTINENCE OF URINE IN CHILDREN

This condition is generally nocturnal, although it may in some instances occur in the daytime, and is one of the most troublesome conditions to treat. No treatment will be successful until the cause of the trouble in the individual case is discovered. These causes may be enumerated as: a long or adherent prepuce in a boy; an adherent clitoris or a vaginitis in a girl; rectal irritations; pinworms or other para-

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142. Zobel, A. J.: Genito-Urinary Symptoms Arising from Anal, Rectal and Colonic Diseases, and Vice Versa, The Journal, A. M. A., Feb. 12, 1916, p. 496.

sites; irritation of the skin in the genital regions; too acid or prolonged alkaline urine; bacilluria; irritations from gravel or from a calculus; bladder irritability; cystitis and pyelitis. All localized skin irritations and inflammations may be either a cause or a consequence of the condition. Indirect causes are digestive disturbances, overloading the little patient with rich, nitrogenous foods, or with too much water at night; disturbances caused by adenoids and enlarged tonsils, or more general disturbances, such as chorea or an unsuspected nocturnal epilepsy.

This nocturnal enuresis is more frequent in boys than in girls, and may continue into childhood, even after the child has been educated to retain the urine.

This affliction may cease suddenly, without any treatment whatever, or it may persist well into youth, and may even occur in maturity. Many of these cases become cured at puberty, if not before, probably because of the normal development of the genito-urinary organs, and hence improved functioning or stronger sphincter of the bladder.

The treatment consists of removing, if possible, all or any of the causes suggested, a proper regulation of the diet, and prohibition of coffee and tea, and of large amounts of even the simplest drinks after the evening meal. Examination of the urine may suggest treatment devoted to disturbances it reveals, such as potassium citrate for hyperacidity, hexamethylenamin in bacilluria, phenyl salicylate when the urine is too alkaline or dilute hydrochloric acid, after meals. If for any reason hexamethylenamin causes irritation or is not advisable, phenyl salicylate in small doses (and all dosage carefully arranged according to the age of the patient) is sometimes successful in curing a bacilluria or an increased secretion of mucus from the bladder; it may also be curative in a mild cystitis. A temporary milk and cereal diet may be effective.

If these treatments fail in a child who has no assignable excuse for such incontinence, atropin may be given to stop the irritability of the neck of the bladder. It should be given at night, and the dose for a child aged 5 years is from $\frac{1}{400}$ to $\frac{1}{300}$ grain, given one or two hours before bedtime.

Sometimes the fluidextract of ergot, given in 20 drop doses to a child aged 5 years, two or three times a day (as it seems to be a stimulant to smooth muscle fiber) may aid in increasing the power of the sphincter.

Hindering a child from lying on his back by a knot tied in the sacral region, or by elevating the foot of the bed, may prevent the urine from as readily forcing itself into the posterior urethra and causing involuntary urination.

At times the passage of cold sounds seems to stimulate the sphincter of the bladder to better tone. Electricity, with the electrode placed so as to cause the sphincter to contract, has frequently been tried, sometimes with considerable success, the faradic current being the most valuable. Various electrodes are made especially for this purpose, for insertion into the urethra or vagina or into the rectum, with a large pad for the other electrode placed over the pubis.

Sometimes this troublesome condition is cured immediately and permanently by freeing adhesions of a prepuce or of a clitoris.

ACUTE CYSTITIS

The cause of this condition should be sought, and if possible, removed, but it may occur without apparent cause.

The treatment is rest in bed, a milk diet, the administration of alkalies in sufficient amount to cause alkalinity of the urine, and just enough morphin or belladonna to stop unendurable pain. This is not often necessary. Sometimes hot sitz baths are of value. In simple acute cystitis such treatment is generally curative in a few days.

CHRONIC CYSTITIS

This is a surgical condition with some tangible cause, and the treatment primarily is of that cause. The condition is frequently not cured without more or less washing out of the bladder.

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